STUDY ON ETIOLOGY, CLINICAL PROFILE, MANAGEMENT, AND FOLLOW UP OF SEIZURES IN CHILDREN IN A TERTIARY CARE CENTRE IN THENI

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CERTIFICATE

This is to certify that the dissertation entitled **"STUDY ON ETIOLOGY, CLINICAL PROFILE, MANAGEMENT, AND FOLLOW UP OF SEIZURES IN CHILDREN IN A TERTIARY CARE CENTRE IN THENI"** submitted by **Dr.JAI PRAKSH** to the Faculty of Paediatrics, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.D. Degree Branch VII (Paediatrics) is a bonafide research work carried out by him under our direct supervision and guidance.

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CONTENTS

ABBREVIATIONS

SD - seizure disorder

SE - status epilepticus

NCSE - non convulsive status epilepticus

LZP - lorazepam DZP - diazepam

MDZ - midazolam

FP - fos phenytoin

RSE - resistant status epilepticus

SFSD - simple febrile seizure disorder

IE - intractable epilepsy

SMR - sexually maturity rate

LAMA - left against medical advice

ILAE - international league against epilepsy

NIH - national institute of health

MTS - mesial temporal sclerosis

GEFS+- genetic syndrome of generalised epilepsy with febrile seizure plus

FS - febrile seizure

ATFSD- atypical febrile seizure disorder

CSE - convulsive status epilepticus

PCSE - partial complex status epilepticus

ASE - absent status epilepticus

HH - hemiconvulsive hemiplegia

HHE - hemiconvulsive hemiplegia epilepsy

CMRO2- cerebral metabolic rate of oxygen

IPPV - intermittent positive pressure ventilation

AEDs - anti epileptic drugs

INTRODUCTION

Acute seizures are a common neurological symptom in sick children. In patients with fever, they include febrile seizures [1,2], acute symptomatic seizures (e.g. in a child with pyogenic meningitis)[3] or initial seizures in a child with epilepsy or epilepsy syndrome[2].Worldwide, febrile seizures are the most common type of acute seizures in children[4]. Most are associated with infections and have a good outcome [5]. In tropical countries, febrile seizures are common but the prevalence of acute symptomatic seizures (which have a poorer outcome) may be higher than Western countries [6-8]. The incidence of both acute seizures and febrile status epileptics is higher[2,9] and the outcome is worse since the etiology is different[6,8,10,11]. Acute seizures are therefore a major risk factor for neurological and cognitive impairment [12-14] and for the development of epilepsy [15-17] in children living in these regions. The incidence is highest in children less than 3 years of age, with a decreasing frequency in older children [18]. Seizures account for about 1% of all emergency department visits, and about 2% of visits of children's hospital emergency department visits [19]. In most of the studies, febrile seizures were reported to be the most common type seen in the pediatric population and account for the majority of seizures seen in children younger than 5 years of age [19-20]. Central nervous system (CNS) infections are the main cause of seizures and

acquired epilepsy in the developing world [20, 21]. Geographical variations determine the common causes in a particular region. Acute seizures are common in meningitis, viral encephalitis and neurocysticercosis and in most cases are associated with increased mortality and morbidity, including subsequent epilepsy [22-25]. The standardized mortality rate (SMR) in patients with a newly diagnosed unprovoked seizure ranges from 2.5 to 4.1 according to the study population and design. The SMR is highest in the youngest patients and in those with symptomatic seizure [26]. In most children with newly diagnosed epilepsy, the long-term prognosis of epilepsy is favorable, and in particular, patients with idiopathic etiology will eventually reach remission [27]. There are limited studies on causes and outcome of acute episode of seizure in developing countries. Most studies had done so far have focused on epilepsy and clinical seizure types [28, 29]. In this study, we therefore analyzed the prevalence of various etiologies, the clinical spectrum of seizure disorders and primary outcome of children admitted with acute seizure disorder. It should be noted that in a developing country like India seizure disorder is a major factor of child hood deaths. Most of them are correctable and mortality occurs mostly due to delay while reporting, (leading to SE and death), proper investigations and treatment or poor compliance and follow up.

DEFINITIONS:

EPILEPSIES:- A group of disorders of CNS manifested by paroxysmal cerebral dysarthythmia, manifesting as brief episodes of loss or disturbance of consciousness, with or without characteristic body movements, sensory or psychiatric phenomenon.

Epilepsies have been classified variously; major types are described below.

I. Generalised seizures

1. Generalised tonic-clonic seizures (GTCS, major epilepsy, grand mal): commonest, lasts 1–2 min. Usual sequence is aura—cry—unconsciousness—tonic spasm of all body muscles—clonic jerking followed by prolonged sleep and depression of all CNS functions.

2. Absence seizures (minor epilepsy, petit mal):

prevalent in children,

lasts about 1/2 min.

Momentary loss of consciousness, patient apparently freezes and stares in one direction, no muscular component or little bilateral jerking. EEG shows characteristic 3 cycles per second spike and wave pattern.

3. Atonic seizures (Akinetic epilepsy): Unconsciousness with relaxation of all muscles due to excessive inhibitory discharges.

Patient may fall.

4. Myoclonic seizures :

Shock-like momentary contraction of muscles of a limb or the whole body.

5. Infantile spasms (Hypsarrhythmia) :

Seen in infants.

Probably not a form of epilepsy.

Intermittent muscle spasm and progressive mental deterioration.

Diffuse changes in the interseizure EEG are noted.

II. Partial seizures

1. **Simple partial seizures (SPS, cortical focal epilepsy):**

lasts 1/2–1 min.

Convulsions are confined to a group of muscles or localized sensory disturbance depending on the area of cortex involved in the seizure, without loss of consciousness.

2. Complex partial seizures (CPS, temporal lobe epilepsy, psychomotor):

Attacks of bizarre and confused behaviour and purposeless movements, emotional changes lasting 1–2 min along with impairment of consciousness.

An aura often precedes.

The seizure focus is located in the temporal lobe.

2. Simple partial or complex partial seizures secondarily generalized :

The partial seizure occurs first and evolves into generalized tonic-clonic seizures with loss of consciousness.

Most of the cases are primary (idiopathic), some may be secondary to trauma/surgery on head, intracranial tumour, tuberculoma, cysticercosis, cerebral ischaemia, etc. Treatment is symptomatic.

Febrile seizures

Comes under special category defined as:-

(a) seizures occurring between age 6 to 60 months.

(b) temperature of 38.c or higher.

(c) not a result of CNS infection or any metabolic abnormality.

(d) in the absence of a history of prior afebrile seizure.

Divided into 2 types:-

(A)Simple febrile seizures:-

primarily generalized,

mostly tonic clonic,

associated with fever,

lasting for a maximum of 15 minutes,

not recurrent within a 24 hrs period.

(B)Complex febrile seizures:-

seizures associated with fever,

mostly focal,

prolonged(>15 mins),

and or recurrs within 24 hrs.

(c)Febrile status epilepticus :-

febrile seizures lasting >30 minutes.

Incidence:-

- Between 2% and 5% of neurologically healthy infants and children experience at least 1 episode, usually simple febrile seizure, respectively.
- Complex febrile seizure has an approx 2 fold long term increase in mortality as compared to general population over 2 yrs.
- Recurrent simple febrile seizure do not damage the brain.
- Febrile seizure recurr in approx of 30% of those experiencing a first episode, in 50% after 2 or more episode, and in 50 % in infants <1yrs old at febrile seizure onset.

About 15% of children with epilepsy have had febrile seizures, only 2-7% of children who experience febrile seizures proceed to develop epilepsy later in life.

- 2 to 4 % of children below 5 yrs of age.
- 10.3% in a study from south India.

- Prevalence is variable with 2.27 per 1000 in a north India study and 3.28- 5.71 per 1000 in south India.

Age of first attack:- in 50% of children the first

attack occur in 2nd year of life and 90% in 3rd year.

Sex :- some studies find higher incidence in boys while other had equal incidence in both boys and girls.

- Family history:- 25-40% with FS have a family history of FS.
- If one parent has FS, the risk in offspring is 10-25%, one sibling leads to 10% risk and one parent plus one sib gives rise to 50% risk of FS.

There are two definition regarding febrile seizures.

(1)NIH- an event in infancy and childhood usually occurring b/w 3months and 5 yrs of age associated with fever but without evidence of intra cranial infection or defined cause of seizure.

(2) ILAE:- A seizure occurring in the childhood after 1 month of age, associated with a febrile illness not caused by infection of the CNS , without previous neonatal seizures or a previous unprovoked seizure, and not meeting criteria for other acute symptomatic seizures.

RECURRENCE:-

- 35% of all children will have recurrence after first FS.
- >50% within 12 months & >90% within 24 months.

RISK FACTORS FOR RECURRENCE:-

(A) FS:-

- Age of onset <12 month
- Complex FS
- Family history in 1st degree relative
- FS with low fever
- FS with short duration b/w onset of fever & FS

(B) Epilepsy following FS:-

- Developmental or neurological abnormalities before 1st seizure
- Complex FS
- Family history of FS in 1st degree relative

RISK OF EPILEPSY:-

- Overall risk following FS is 2-5%
- Approx 13-19% children who develop epilepsy have had previous FS
- Risk of epilepsy with no risk factors is 0.9%, with two or more risk factor is 2%
- If FS is prolonged approx 9.4% of children may develop epilepsy

RISK OF MTS:-

- There is strong association between development of refractory MTS and complex FS (especially prolonged or focal)
- But it is not clear whether the prolonged FS were cause of MTS, or whether preexisting MTS predisposes child to have prolonged FS.
- Mental and neurological development remains normal if they are normal before onset of FS
- There is no increased risk or incidence of mortality in children with FS, including febrile SE and excluding infection of CNS

GEFS +:-

- Genetic syndrome of generalized epilepsy with febrile seizures plus
- This term means FS continuing beyond age of 6 years when typically the FS should disappear
- The syndrome may manifest with just an isolated FS , which is the mildest manifestation , simple FS, FS+ or seizure with other associated afebrile seizure which could be myoclonic atonic or absence seizure .
- Most severe manifestations are DRAVET SYNDROME and myoclonic astatic epilepsy
- Except for these two syndromes the prognosis is generally good and the febrile or afebrile seizures remit over time.

STATUS EPILEPTICUS

Status Epilepticus (SE) is one of the most critical medical emergencies that may result in significant morbidity and mortality if not addressed in a timely and effective manner(29). The approach in generalized convulsive SE is modified by changing concepts regard the definition of SE and studies justifying more aggressive treatment, with earlier intervention started prior to arrival to hospital. Currently SE has 1/5 the morbidity and 1/3 the mortality of pre -1970(30). But still mortality is around 11-53%(31,32,33). Improvements reflect studies, retrospective data, changing definition of SE (from >60 minutes to > 5 minutes). But most important factor is improved care. Although most seizures in children stop prior to arrival at a hospital, an estimated 60,000 US children are treated each year for SE. 1/3 of the episodes will be initial event in a patient with new onset epilepsy and an additional third occur in children with established epilepsy. Up to 70% of children with epilepsy beginning before age 1 will experience as episode of SE in their life time. Incidence is about 50000-2.5 Lakhs times/year in US. 21% were <1year and 64% were <5 years. <50% of SE has h/o epilepsy. 15% of the epileptics will have SE at one time. 10% of the epileptics will present with SE at I time itself(30).

PATHOPHYSIOLOGY

SE is an emergency and must be treated immediately. Cardio respiratory dysfunction, hyperthermia, metabolic derangements, irreversible neuronal injury can develop as a consequence of prolonged SE(37). CNS injury can occur even when the patient is paralyzed with neuromuscular blockade but continues to have electrographic seizures(34).

SEIZURE INITIATION AND PROLONGATION:-

The mechanism of start & end of seizure is unknown, but it is proposed that seizure initiation is caused by an imbalance between excitatory and inhibitory neurotransmission, causing initiation of abnormal neural impulses. Excitatory synapses matures earlier than inhibitory which, along with an increase in the susceptibility of excitatory neurotransmitter receptors, increases the likelihood that an imbalance between excitation-inhibition may occur.

The underdeveloped cerebral cortex has a high synaptic density at around 2 months of age and this may led to the development of excessive synchrony of neural groups. The excitatory amino acid neurotransmitter glutamate increases at the site of the seizure at the beginning of seizural activity in adults with temporal lobe epilepsy & is thought that the same may happen at the onset of generalized seizures. Inhibitory neurotransmitters such

as GABA increase later at the seizural site and disarrange the balance between excitation and inhibition. Other mechanisms of inhibitory receptor modulation, example adenosine receptor agonism, can also contribute to end of seizure. Thus the increased incidence of seizure disorder in childhood is probably caused by a combination of increased seizure susceptibility and decreased ability for an adequate inhibitory response. The spike and wave discharges occur when cortical neurons will be either depolarized (spike) or hyper polarized (wave).

PHYSIOLOGY:-

About 70-80% of CSE throughout all age groups will have a focal onset but may be secondarily generalized. A predictable change of sequence in the EEG has been shown in humans and in animal models. CSE starts with localized activity of epilepsy followed by isolated generalized bursts of seizural activity with a normal EEG in between the attacks. If the patient does not regain consciousness, then clinical criteria for CSE is achieved. The isolated ictal discharges merges and become a continuous after 30 minutes. They then fragment and became interspersed with flat periods. Finally, a periodic epileptiform discharge occurs, which may reflect underlying metabolic failure.(35).

MOTOR RESPONSE :-

The motor response associated with CSE follow a similar pattern of the EEG. Recurrent seizures merges into continuous motor activity, followed by fragmented motor activity and myoclonus. If the seizural activity persists, then electromechanical dissociation occurs. The prognosis of a good neurological outcome decreases as the seizure persists beyond this phase.

ROLE OF EXCITATORY AMINO ACIDS :-

They are important in causing structural brain lesion secondary to prolonged seizure. MTS is the most common brain lesion following prolonged seizure and may result from excitotoxicity. Most efforts were made to know the effects of glutamate. Prolonged seizures can cause hippocampal damage and results in neural loss in the amygdala, neocortex, hippocampus, cerebellum & thalamus. Bilateral damage of hippocampus may occur even with unilateral stimulation with excitatory neuro transmitters.

Prolonged seizures induces the production of heat shock proteins in many areas of brain. These heat shock proteins protects the brain against the stressful stimuli, which will be potentially harming to neurons. The result is that prolonged seizures may need to occur in epilepsy human patients for MTS to develop, and once it has developed further episodes of prolonged seizure may not worsen the MTS(36).

GLUTAMATE CAUSES CELL DEATH:-

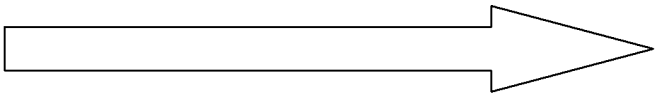
Excess extra cellular glutamate results in cell death by leading to necrosis, gene determined cell death, or both. The primary receptor involved is the NMDA receptor, but other glutamate receptors can also be involved. The NMDA is an inotropic receptor. Influx of calcium through the ionophore occurs due to the locking of glutamate and glycine or D-serine to the correct site on the receptor. High intracellular calcium concentrations result in the activation of large number of calcium dependent processes such as activation of protein kinase C, nitric oxide and free radical formation, activation of phospholipase A2 and activation of protease calpain I.

Pathophysiological consequences following seizure is extensively studied in animal models. There is no deficit in brain energy state until later around 1 hour, when parenchymal oxygen falls. Then brain damage occur(37). Several investigations have shown that seizures become more difficult to stop and the chances of neuronal injury increase when seizures persist beyond a transitional period that varies between 20 and 60 minutes in animals during constant seizure activity. Treatment in children should be directed to supporting vital functions and to controlling the convulsions as expeditiously as possible, because the precise transitional period in humans is not known.

PROLONGED SEIZURE

Temporary systemic change	Life threatening systemic change	Death

Duration of seizure



SYSTEMIC AND CENTRAL PHYSIOLOGY

The systemic effects of seizure is initially dominated by the body's attempt to maintain homeostasis. Bp & CVP increase, blood sugar increases, and tachycardia occurs. It may also results in dyselectrolytemia and hyperthermia. Cerebral blood flow, blood sugar, and oxygen consumption increase in the early phases of a seizure to maintain cerebral homeostasis.

After 30 minutes, homeostatic failure occurs and the patient will need systemic supports. Cerebral blood flow, brain sugar, and parenchymal oxygenation decrease and causes cell damage associated with SE. Respiratory and metabolic acidosis, dyselectrolytemia(for example, hyperkalemia), hyper or hypothermia, and rhabdomyolysis may all occur. Drugs with depressant cardio respiratory actions (for example, benzodiazepines and barbiturates) may worsen the systemic complications of SE(37).

GLUCOSE

Initially hyperglycemia and in phase II become hypoglycemic.

• Hyperdynamic phase – Hyperglycemia

⇩

Exhaustion phase – Hypoglycemia develops –

⇩

Hypoglycemia appears in presence of hypoxia

⇩

Neuronal damage ensues

HYPOXIA

Associated with SE is multi factorial. Impaired mechanical ventilation secondary to tonic clonic activity, increased salivation and tracheo bronchial secretions obstructing the airway, increased O2 consumption resulting from the seizure, drugs to terminate SE are respiratory depressants that cause hypoventilation are some of the factors.

ACIDOSIS

Acidosis of SE is of both respiratory and metabolic in origin as seizure activity results in increased metabolic needs unmet by tissue oxygenation and perfusion, causing lactic acidosis.

• Hyperdynamic phase – CBF meets CMRO2

⇩

• Exhaustion phase – CBF drops as hypotension sets in –

⇩

Autoregulation exhausted –

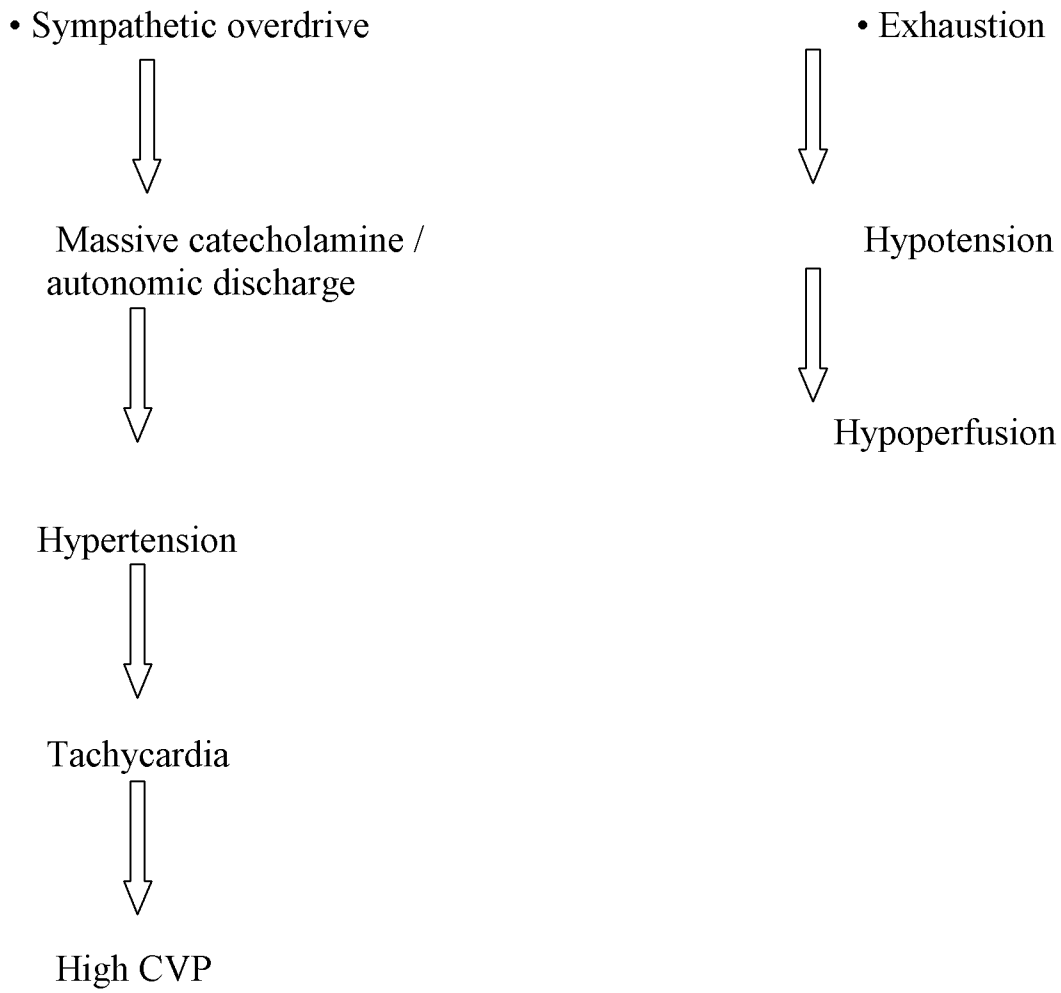
⇩

Neuronal damage ensues

HEMODYAMICS

In first 30 minutes of seizure activity, catecholamine release results in an increase in heart rate, blood pressure, central venous pressure, cerebral blood flow and serum glucose. After 30 minutes of GTCS, Blood pressure begins to drop and cerebral blood flow although still increased above base line, drops to the point where it may be unable to supply adequate substrate and oxygen to meet increased cerebral metabolic demands. This results in impaired cortical oxygenation (37). Other effects are ↑ CPK, Myoglobinuria, ATN, trauma, tendon rupture. Seizure duration greater than 1 hour, especially with hypoxia, has been associated with permanent neurologic injury.

Hemodynamics



ALTERED MENTAL STATUS /NCSE

Convulsions are easily identified as the source of altered mental status if typical tonic clonic movements are witnessed. However, children may also present in a post ictal state, without a clear history of a seizure, thus making the diagnosis more difficult to determine. Further more, seizures may be followed by a period of transient paralysis (Todd's paralysis) that is often present on one side of the body. This may lead to the clinician to suspect a structural etiology rather than seizures. NCSE should be considered in comatose children without signs of seizures activity, an EEG, will help to diagnose this condition(39).

NCSE

If the patient stops overt convulsions yet remains comatose an EEG should be performed to rule out ongoing S.E. Up to 20% of children with SE have non - convulsive SE after tonic - clonic SE(40). Neurologic signs after termination of SE are common: pupillary changes, abnormal tone, abnormal Babinski reflex, posturing, clonus, may be asymmetrical. They should not be misinterpreted as NCSE.

NCSE is commonly diagnosed in children, where as acute delirium status is frequent in adults ED diagnoses. NCSE is usually entertained only when the child is unresponsive and rigid or has a known seizure disorder. But PCSE and ASE both can present with subtle findings erroneously ascribed to

other etiologies and may occur in patients with out a known seizure history(41).

NCSE accounts for almost 20% of SE(41). PCSE has been associated with neuronal damage and stroke(42) and is more likely caused by primary pathology of the cortex such as an infection or bleed. In contrast ASE probably has a different and less harmful origin, seemingly resulting from vacillating thalamo cortical excitation and excessive synchronous neuronal inhibition, which could explain the absence of tissue injury following its resolutions(42). In PCSE, as in GTCS excessive excitatory neuro transmitter release leads to depolarization and increased intracellular calcium. When GABA inhibition is overwhelmed, calcium triggered proteases and lipases leads to cell injury and death(42).

REFRACTORY STATUS EPILEPTICUS

RSE is defined as seizures that do not controlled with adequate does of BZD, Phenytoin or Phenobarbital and require more aggressive treatment(43). Complications are more and out come poor in RSE. Continuous IV infusions of anesthetic doses of midazolam, propofol, or barbiturates are the most useful treatments(44). Children with prior, new, progressive CNS injury are more prone to have RSE.

TYPES OF SE(35)

Convulsive SE (CSE)

GCSE/ GTCS (primary)

Secondary generalized

Multifocal clonic

Hemi convulsive SE

Tonic SE

Clonic SE

Simple PSE/ Epilepsia partia continualis

Non convulsive SE (Subtle SE)

Neurological EMD (Electrical SE)

PCSE

ASE

Myoclonic SE +/- salaam attacks

MEDICAL COMPLICATIONS OF STATUS EPILEPTICUS

STATUS INDUCED

Epilepsy subsequently (77%)

Hemi convulsion Hemiplegia (HH)

Hemi convulsion Hemiplegia Epilepsy (HHE)-rare now

INTER ICTAL COMA

CUMULATIVE ANOXIA

Cerebral and systemic

CARDIO VASCULAR COMPLICATIONS

Tachycardia, Bradycardia

Cardiac arrest

Hypertension, Hypotension

Cardiac failure,

Cardiogenic shock

RESPIRATORY SYSTEM FAILURE

Apnea

Chyne stokes breathing

Bradypnea,

Tachypnea

Neurogenic pulmonary edema

Aspiration pneumonia

Respiratory acidosis

Cyanosis

RENAL FAILURE

Oliguria

Uremia

ATN

Rhabdomyolysis

AUTONOMIC SYSTEM DISTURBENCE

Hyperpyrexia

Excessive sweating, vomiting

Hyper secretions (salivary, tracheo bronchial) Airway obstruction

METABOLIC AND BIOCHEMICAL ABNORMALITIES

Acidosis (metabolic, lactate),

Anoxemia

Hypernatremia, Hyponatremia

Hyperkalemia

Hypoglycemia

Hepatic failure

Dehydration

Acute Pancreatitis

Leucocytosis

INFECTIONS

Pulmonary

Urinary

Skin

OTHERS

Altered auto regulation of CBF

Increased cerebral metabolic rate of O_2 (CMR O_2)

DIVC

MODS

Fractures

Thrombo phlebitis

ETIOLOGY

(1) INFECTIVE:-

- Acquired bacterial meningitis,
- TB meningitis,
- aseptic meningitis,
- encephalitis,
- cerebral malaria,
- tetanus, mumps, encephalopathy, measles encephalopathy and Reye's syndrome.

(2)METABOLIC CAUSES

- Dehydration
- dyselectrolytemia,
- acidosis, alkalosis,
- Hypoglycemia, Hyperglycemia, and inborn errors of metabolism.

(3)SPACE OCCUPYING LESIONS:-

- Neoplasm of brain,
- Brain abscess,
- Tuberculoma,
- Cysticercosis.

(4)VASCULAR:-

- Arteriovenous malformations,
- Intracranial Thrombosis (or) Haemorrhage,
- Consumptive Coagulopathies. Congenital Malformations, Migration defects, trauma.

(5)MISCELLANOUS CAUSES:-

- Anoxic / Hypoxic Ischemic Encephalopathy,

- Residua of birth trauma and birth asphyxia,
- heat stroke,
- toxic encephalopathy,
- lead encephalopathy,
- Hypertensive encephalopathy,
- breath holding spells,
- grey matter degeneration,
- storage disorders.

(6)DRUGS AND POISONS

- Toxic doses of Phenothiazine, Salicylate, Diphenylhydantoin, Strychnine, Carbon monoxide, dapsone.

APPROACH TO A CONVULSING CHILD

(1) To stablize the child according to the universal ABCD Protocol (discussed later).

(2) Proper history:- including type of seizure, duration of seizure, no. of episodes, h/o loc, fever, family history, etc

(3) Detailed examination:- of all system, stressing more over CNS(look for any NCM if present), locate signs of CNS infection (if this is the first episode

of seizure, associated with fever or altered consciousness), any signs of metabolic abnormality(dehydration, hypoglycemia , etc)

(4) Investigations:-

- CBC
- Urine routine
- Stool routine
- Mx
- Chest Skiagram
- Blood culture and peripheral smear

- To rule out metabolic causes
  - Blood sugar
  - Calcium
  - Magnesium
  - Electrolytes
  - Urea
  - Creatinine.

(4)FUNDUS EXAMINATION:-

This should be done in all babies as it gives clues regarding blurring of disc margins, tortuosity of retinal blood vessels etc.

(5)EEG

Not necessary in a case of 1st febrile seizures

Indication – recurrent / atypical febrile seizure

(6)IMAGING –

CT/MRI/MRA or MR SPECTROSCOPY

to rule out CNS infections, SOL or any major structural deformity.

(7) CSF Examination:-

(a) features of meningism.

(b) the seizure is a complex FS.

(c) the child is drowsy, irritable or systemically ill.

(d) child <18 months(probably) and definitely if child is <12 months1st episode of febrile convulsions < 12 months.

AAP GUIDELINES FOR LP:-

Complex fs	Simple fs (previously neurologically normal child)		SIMPLE FS (previously neurologically abnormal)
DEFINITE LP	DEFINITE LP	OPTIONAL LP	CLINICAL JUDGEMENT
ANY AGE	ANY AGE : FEATURES OF MENINGITIS OR CNS INFECTION	ANY AGE : PRETREATMENT WITH ANTIBIOTIC <12 Months : UNIMMUNISED WITH H.influenze AND S.pneumonia OR IMMUNIZATION STATUS NOT CLEAR	

MANAGEMENT:-

Longer the duration of seizures, greater the risk of complications, so, every attempt should be made to control epileptic activity (clinically and electrically) as soon as possible. Seizure is a medical emergency that requires an organized and skillful approach to minimize the associated mortality and morbidity. CSE is one of the most critical neuroemergencies and every physician confronted with these patients in the emergency department should have a protocol, how to terminate seizure. Therapy must be aggressive because neuronal excitability can be reversed only early in the course and quick intervention may decrease the risk of seizures generated neuronal damage.

The longer Seizure persists, the lower is the likelihood of spontaneous cessation, the harder it is to control and the higher is the risk of morbidity and mortality(44). SE is a condition which most likely will not terminate rapidly or spontaneously and requires prompt intervention(45). Primary aim is to control and abort the seizure as the duration of seizure activity is directionally proportional to immediate mortality and later morbidity. Goal of treatment is the cessation of both clinical and electrical seizures. A time framed protocol is essential in the management of SE in ER.

Goal of management is, to maintain adequate vital function and oxygenation, to terminate seizure activity as quickly as possible, to evaluate

and treat the underlying cause of SE. Ventilation is very important. Use of AEDs to stop seizures and to stop respiration for intubation is better than giving neuromuscular blockade alone. Use correct and adequate AEDs doses. Epileptics and non-epileptics in status require the same drug doses. It should be remembered that outcome is determined by etiology, age, duration and treatment. We can affect only the treatment. Most easily missed causes of status epilepticus are bacterial meningitis, encephalitis, abuse/unsuspected trauma, drug ingestion.

Priority is the management of ABCs along with rapid termination of seizure activity. BZDs are the initial drug of choice in terminating SE. LZP, DZP, MDZ are the recommended drugs. Though DZP and LZP are equally effective in controlling seizures LZP is preferred because if it's longer duration of action(46,47). If DZP is given it should be followed by a long acting drug AEDs such as phenytoin to prevent recurrences (20mg/kg, followed by 10mg/kg, total of 30 mg/kg). MDZ has no more advantage over DZP or LZP in efficacy, onset and duration but can be given as IM injection. So, ideal for pre hospital therapy and it can be given if IV access could not be obtained(48,49). Intramuscular MDZ is given as 0.2 mg/kg and it is an aqueous solution and rapidly absorbed, anticonvulsant effect begins after 2 minutes. Intramuscular LZP also can be given, but lacks water solubility, thus later onset than MDZ.

Phenytoin is given as 20 mg/kg I.V. over 20 min, then if needed 10 mg / kg IV infusion over 20 min. Its pH is 12, extravasations cause severe tissue injury. Onset is 10-30 min and may cause hypotension, arrhythmias, ventricular standstill(50- 58) but cost-effective.

FP is a newer pro drug of phenytoin. Given as 20 mg PE/kg IV over 5-7 min, PE = phenytoin equivalent, pH 8.6, extravasations are well tolerated. Onset is 5-10 min, may cause hypotension, but less and it is expensive(59-68). The main difference between phenytoin and FP in children is the pH. FP will not cause the severe tissue damage seen with phenytoin in case of infiltration. If in doubt serum level free phenytoin should be measured. Phenytoin is largely protein bound. (> 90%, varies with serum protein concentration).Free phenytoin = active phenytoin (anticonvulsant and toxic effects). Toxicity is more likely with hypoalbuminemia (usually if < 2 g/dl). Therapeutic levels: Total phenytoin: 10 - 20 mcg/ml, Free phenytoin: 0.8 - 1.6 mcg/ml

In RSE, MDZ given as 0.2mg/kg IV bolus followed by 0.75 to 10 mcg/kg/min infusion (69-73). Propofol is given as 1to2 mg/kg loading followed by 2 to 10 mg/kg/hr (74-78). Both drugs have the substantial advantage over barbiturates of rapid clearance and MDZ has less pronounced hypotensive effects. Midazolam infusion is typically maintained for 12 to 24 hours and is then withdrawn gradually while the patient is observed for

clinical and EEG evidence of seizure termination over next 48 to 72hrs. If seizure continues the therapy should be resumed for prolonged periods. Midazolam may be associated with tachyphylaxis leading to the need for exceedingly high doses. Propofol is a pro convulsant. Seizure during induction and termination was reported but, these effects in the management of SE are unknown.

Thiopental and pentobarbital are potent anti seizure drugs that have potential though unproved cerebral protective effects in the management of SE(79-87). In adequate doses these drugs will always control seizures, but severe hypotension requiring blood pressure controlling measures limits their safety. But in case of hospital set up like ours can be tried under close observation of the patient's vitals. So, these are reserved for failed midazolam / propofol. In resistant cases, Inj. Sodium valproate 20-40 mg/kg IV bolus as infusion may be used(82-106).

EEG

In a child with a new onset of seizures, an EEG may help to differentiate ictal from non ictal events, to determine seizures type or epilepsy syndrome and to better define the risk for recurrence. For most children it is not necessary to perform the EEG as part of the initial emergency department evaluation. It is performed shortly after the seizure (<48 hours) the EEG may show diffuse post ictal slowing without prognostic significance(107,108,109).

Among children with persistent altered mental status after a seizure an emergent EEG is helpful to identify subtle or NCSE(110) .

NEURO-IMAGING: CT Brain may be necessary to evaluate safety of LP and to rule out hemorrhage or large mass lesions. MRI will almost always be performed later, even if CT is normal.

LP : LP should be done if SE presented in febrile children. And it should be done only after stabilizing the child not at arrival.

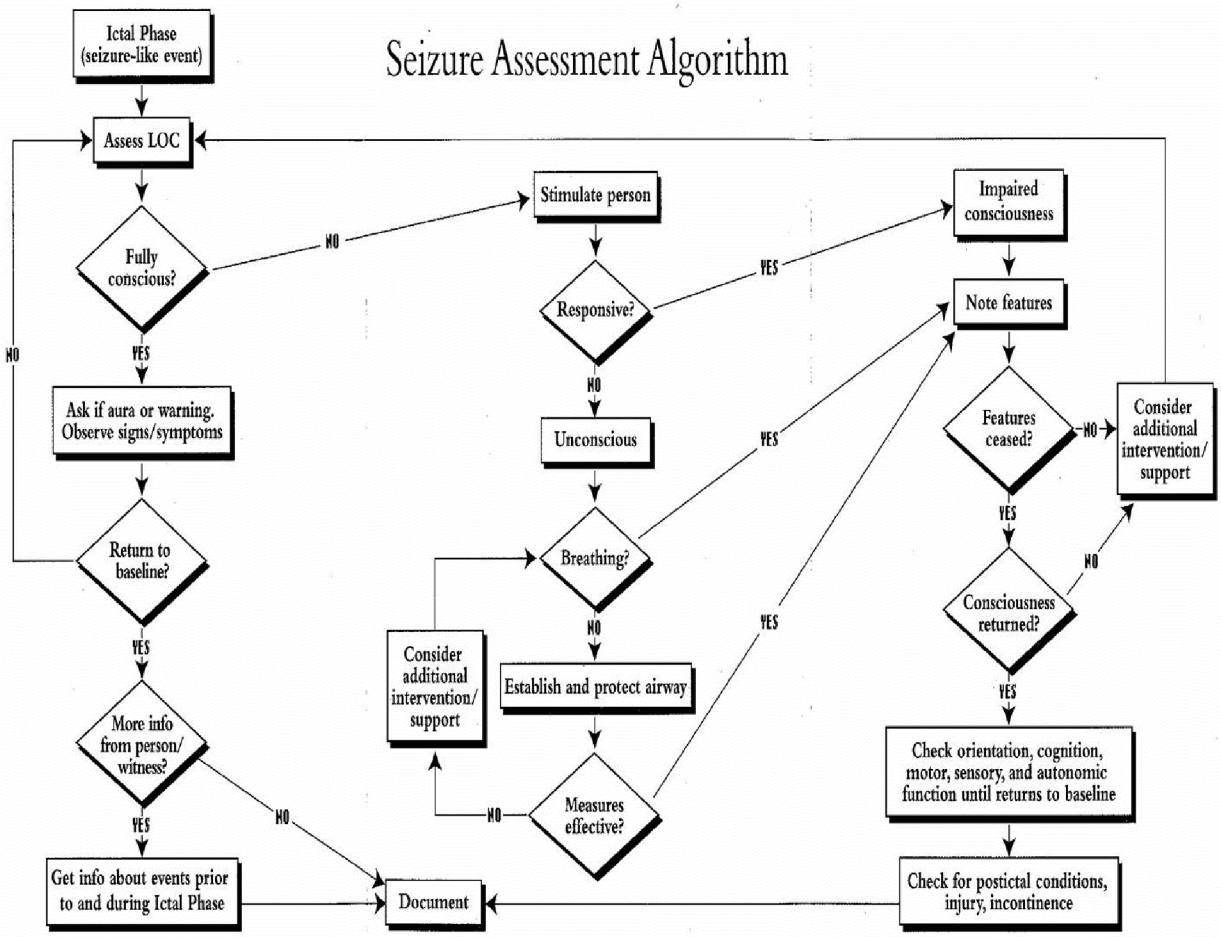
OUTCOME

Outcome is determined by, Etiology, Age, Duration, Treatment (31,32,33). Ultimately mortality related to, damage to the CNS caused by the acute insult precipitating SE, systemic stress from SE (major cause because of anoxia, acidosis, shock), injury from repetitive epileptic discharges within the CNS (minor but cognitive impairment later). Mortality increases from 3% to 32% if the duration of seizures becomes >1 hour.

Etiology	Risk of Epilepsy	Risk of Epilepsy	Chronic AED Rx
Idiopathic	0%	3%	Absent
Remote symptomatic	++	50%	Present
Febrile	+	+	Absent
Acute Symptomatic	15=30%	+	Present
Progressive	++	50%	present

Normal children and children with febrile status have a favorable prognosis. Improved outcome is a result of timely and approximate evaluation and treatment. Most favorable for patient who respond to first line agents, but the underlying cause of seizure, determine the outcome. Cognitive function may be impaired (particularly memory) in patients with prolonged seizure & is more common when significant hypoxemia (aspiration) is present. Outcome may be worse when S.E. is managed inappropriately. Most common mistakes are, inadequate dosing, failure to order maintenance therapy , late reporting to hospital inadequate dose of the discharge drugs results in recurrence. AED should be continued particularly if a structural lesion resulted in S.E.

TREATMENT CATALOGUE



REVIEW OF LITERATURE

Prakash Poudel et al,Prince Parakhet al, Kayur Mehta et al, a study done in Nepal for etiology & outcome of seizure disorders in children , showed following results (20)

Study included 308 (age one month to 20 years) children. Median age at first seizure was 39 (inter quartile range 12-96) months. History of status epilepticus was present in 26.0%. Cause of seizure was known in 44.2%. Seizure was generalized in 79.2%, partial in 14.0% and unclassified in 6.8%. Common causes of seizure were – birth asphyxia (12.3%), neurocysticercosis (8.8%), sequel of nervous system infection (6.5%) and structural brain abnormalities (7.1%). Neurological examination, electroencephalography and computed tomography (CT) were abnormal in 24.4%, 70.5% and 27.9% cases respectively. Seizure control was achieved in 79.3% and by monotherapy in 85.0 % cases. Seizure control with single drug, seizure without recurrence and idiopathic seizure were associated with favourable outcome.

S.Sarvanan et al(34) concluded amongst total of 520 patients admitted for seizures in Meenakshi medical college & research institute, MAHER University, Tamilnadu, India] with 300 (57.7%) males and 220 (42.3 %) females. Among these patients, 268(51.5%) presented with fever and 388 (74.6%) of children were less than 6 years of age. Generalized seizures were

the most common seizure type (50.2%). Febrile seizures (36.5%), seizure disorder (33.2%), symptomatic seizures (20%) and space occupying lesions were common etiologies. Abnormal brain images were noted in (25%) of 424 patients and most common abnormality in space occupying lesion was neurocysticercosis (4%).

Ramesh Baheti et al, BD Gupta et al, Rajesh Baheti et al(22) concluded amongst 52 children with seizure disorder were included; 26 of them were having partial seizures, while the rest (2 6) were having generalised seizure Abnormal EEG was found in 73% and 76.9% of patients with partial and generalised seizures respectively, while abnormal CT scan was found in 50% of patients with partial seizures and 34.6% of patients with generalised seizures. It was also observed that with increasing abnormalities in EEG, the chance of finding some abnormality in CT scan also increases.

Vrajesh udani et al(81) concluded in a study conducted in Grants medical college & J J group of hospitals in 2005 that incidence of FS in india is similar to other countries & appears to be benign. Benign epilepsies & West syndrome remains under reported. Risk factor for IE includes early age of onset, neurodevelopmental abnormalities & certain seizure types. Perinatal injuries underlie many IE. NCC & Hypoglycemia were found to be most common cause

Shakirullah et al, Niaz Ali et al, Aslam khanet al, Muhammad Nabi et al (69)concluded in a study conducted in Basic Medical Science, Khyber Medical University Peshawar in 2014 concluded that Epilepsy is a neuronal disorder that is observed globally but still it is not explored very well in most parts of the world. This disease is linked to different provocative causes and affects almost all generation, ethnicity and age population. The worldwide prevalence of epilepsy is variable and varied among countries. High prevalence is found in adolescent and early age group population. In North America, Central and South America high prevalence is found in male except in New York, Bolivia, Honduras and Argentina where prevalence is high in female. In Asian countries such as China, India, Turkey and Saudi Arabia the prevalence is high in Male except in Pakistan here prevalence is high in female similarly to European countries where also prevalence is high in female. The prevalence of epilepsy in male and female is variable in African countries. Generalized seizure is high in America, Asia, Europe, and Africa than the other types of epilepsies. Very limited data is available about the incidence of epilepsy especially from low and lower middle income countries. The incidence rate of epilepsy is higher in the developing countries than the industrialized countries. Similarly, the incidence is also higher in male than female. Head injury, birth trauma, cerebrovascular disease, and intracranial infections (neurocysticercosis or meningoencephalitis) and genetic factors are the main causes of epilepsy.

MRITUNJAY KUMAR et al, RASHMI KUMARI et al, NIGAM PRAKASH NARAIN et al (76)conducted a study in Bihar, India, 2008 concluded with Preponderance of male (60%) over female (40%) was observed. Aetiology included Idiopathic (27.14%), remote symptomatic (20%), acute symptomatic (47.14%), febrile (2.86%) and progressive encephalopathy (2.86%) groups. Generalised tonic clonic convulsion (GTC) convulsion was observed in 91.4% of SE patients while 8.6% had partial SE. Eighteen patients (25.7%) had prior history of convulsion whereas 52 patients (74.3%) presented with SE as first episode of convulsion. Mortality rate was 31.4% and acute symptomatic causes were responsible for most of the deaths.

Sheffali Gulati et al, Veena Kalra et al and M.R. Sridhar et al(65) from Department of Pediatrics, All India Institute of Medical Sciences, Ansari Nagar, New Delhi, India, in 1993 concluded with A retrospective study of case records of 451 neuroemergency patients admitted in PICU in a tertiary care center out of which 30 patients had status epilepticus. They were evaluated for their clinical presentation, laboratory parameters, treatment profile and immediate outcome. The age group varied from 1 to 120 months with mean of 56.6+ 46.5 months. Seventeen patients were less than 60 months. Sixteen patients (53.3%) presented with SE as first presentation without prior history of seizure activity. Nine patients died (30%) during hospital course. Seizure duration >45 minutes (p-0.001) and presence of

septic shock (p-0.001) were associated with significantly more mortality. There is a need to abort seizure activity at the earliest and this improves immediate outcome.

Shirley A. Russ et al,Kandyce Larson et al and Neal Halfon et al in 2007 amongst 977 children from Department of Academic Primary Care Pediatrics, Cedars-Sinai Medical Center, Los Angeles, California; University of California U.S ,concluded with(52)

Estimated lifetime prevalence of epilepsy/seizure disorder was 10.2/1000 (95% confidence interval [CI]: 8.7–11.8) or 1%, and of current reported epilepsy/seizure disorder was 6.3/1000 (95% CI: 4.9–7.8). Epilepsy/seizure disorder prevalence was higher in lower-income families and in older, male children. Children with current reported epilepsy/seizure disorder were significantly more likely than those never diagnosed to experience depression (8% vs 2%), anxiety (17% vs 3%), attention-deficit/hyperactivity disorder (23% vs 6%), conduct problems (16% vs 3%), developmental delay (51% vs 3%), autism/autism spectrum disorder (16% vs 1%), and headaches (14% vs 5%) (all $P < .05$). They had greater risk of limitation in ability to do things (relative risk: 9.22; 95% CI: 7.56–11.24), repeating a school grade (relative risk: 2.59; CI: 1.52–4.40), poorer social competence and greater parent aggravation, and were at increased risk of

having unmet medical and mental health needs. Children with prior but not current seizures largely had intermediate risk.

William D. Gaillard et al, Catherine Chiron at al, Helen Cross et al, Simon Harvey et al, Ruben Kuzniecky et al, Lucie Hertz-Pannieret al, and L. Gilbert Vezina et al for the ILAE, Committee for Neuroimaging, Subcommittee for Pediatric The International League Against Epilepsy (ILAE) Subcommittee for Pediatric Neuroimaging New york, U.S in 2009 examined the usefulness of, and indications for, neuroimaging in the evaluation of children with newly diagnosed epilepsy. The study was mainly neuro imaging findings in children with new onset seizure concluded that (44):-

The retrospective and prospective published series with n 30 utilizing computed tomography (CT) and magnetic resonance imaging (MRI) (1.5T) that evaluated children with new-onset seizure(s) were reviewed. Nearly 50% of individual imaging studies in children with localization-related new-onset seizure(s) were reported to be abnormal; 15–20% of imaging studies provided useful information on etiology or and seizure focus, and 2–4% provided information that potentially altered immediate medical management. A significant imaging abnormality in the absence of a history of a localization-related seizure, abnormal neurologic examination, or focal electroencephalography (EEG) is rare. Imaging studies in childhood absence

epilepsy, juvenile absence epilepsy, juvenile myoclonic epilepsy, and benign childhood epilepsy with centrotemporal spikes (BECTS) do not identify significant structural abnormalities. Imaging provides important contributions to establishing etiology, providing prognostic information, and directing treatment in children with recently diagnosed epilepsy. Imaging is recommended when localization-related epilepsy is known or suspected, when the epilepsy classification is in doubt, or when an epilepsy syndrome with remote symptomatic cause is suspected. When available, MRI is preferred to CT because of its superior resolution, versatility, and lack of radiation.

Hui AC, et al (88): Factors of poor outcome, defined as death or morbidity as measured by deterioration in functional status using the Glasgow outcome score were analyzed in a multivariate logistic regression model. There most common causes were CVA, metabolic derangements and anticonvulsant withdrawal in adult population. 26% had worst functional ability, and mortality rate was 26%. Predictors of poor outcome were older age (OR = 1.04, 95% CI 1.01 – 1.07), delay in treatment (OR = 9.73, 95% CI 1.58 – 59.96) and CNS infection 9 OR = 30.27 95% C 3.14-292.19).

Veryty CM et al(86): Outcome of SE & lengthy FSE: Findings of National Cohort Study. OBJCTIVE: To study out come after a lengthy FSE & SE in children. DESIGN: Population based birth cohort study. SETTING:

Child health and education study. (16004 neonatal survivors born in April 1970),USA. 14676 – Information available. OBSERVATION: Clinical information and tests of intellectual performance at 5 years and 10 years after birth.19 had lengthy FSE, 18 had SE. 2SE died, but not due to it. 4/19 FSE (21%) developed seizures. Intellectual performance: 23 normal, 10 abnormal (8 preceding developmental delay, neurologic abnormality).CONCLUSION: Outcome is better that reported from studies, seems determined more by the underlying cause than by seizures themselves.

Mah JK, Mah MW (87): King Khalid national guard hospital, Jeddah, kingdom of SA. Pediatric SE: Perspective from Saudi Arabia. Objective: To assess risk factors and management of SE and non SE seizures. DESIGN: prevalence study of a convenience sample of pediatric seizure admitted from 1992 to 1997. RESULT: Mean age 2year 4month. 43% no prior seizures. 28% (59) of 212 seizures were SE. SE more than non SE in cases with h/o seizure, AED, acute etiology. CONCLUSION: Management of SE in this referral population can be improved by more rapid access to appropriate medical care.

Sahin M et al 99(89): out come of severe RSE in children. Epilepsia 2001 Nov: 42(11)1461-7. OBJECTIVE: out come of RSE in children. Retrospective review of case records between 1992-2000.children's hospital, Boston, Massachusettes,USA. Factors evaluated age, etiology and initial EEG

findings including mortality or return to baseline. RESULTS: 22 patients 4.5 months to 18 years treated for < or = to 146 days. Mortality 7/22(31.8%) related to age, etiology and initial EEG findings. None return to base line. No death in remote symptomatic group.3/4 younger than 3 years died.4/18 older than 3 years died. Focal abnormalities in EEG are associated with less mortality than multifocal or generalized abnormalities. CONCLUSION: High mortality and morbidity for childhood RSE is demonstrated.

Maytal J et al 98(108): In an ongoing study of SE, 193 children with SE of varying causes have been followed up for a mean period of 13.2 months. Of those 97 were recruited prospectively. The patient's ages varies from 1 month to 18 years. With a mean of 5 years. The cause was classified as idiopathic in 46 cases, symptomatic in 45, febrile -46, acute symptomatic - 45, & progressive neurologic in 11. 7 died and new neurologic deficits were found in 17 (9.1%) with 186 survived. All of the deaths and 15/17 sequele occurred in acute and progressive neurologic insults group. Only 2/137 other causes sustained any new deficits ($p<0.001$). Duration of SE affects outcome only with in the acute symptomatic group. Sequele occurred mostly in infants (29%) and 11% in 1-3 years group, 6% in > 3 years. 61 had prior unprovoked seizures (32%)

Kalra Veena et al 97(57): OBJECTIVE: To study the clinical profile immediate outcome and risk factors of SE in Children admitted in PICU.

DESIGN: Retrospective, study of case records. SETTING: AIIMS; PERIOD: 451 Neuro emergencies between Jan 1993 to April 2000.in children in a tertiary care center. –30 had SE. INCLUSION: 30 SE Cases. RESULTS: Age: 1- 120 months. Mean 56.6 ± 46.5 months. 17 patients were < 60 months.16 patients (53.3%) had SE I episode with out prior H/o fits.9 (30%) died during hospital course. Seizure duration >45 min (p.0.001) and presence of septic shock (p-0.001) were associated with significantly more mortality. RESULT: abort seizure as soon as possible.

AIM AND OBJECTIVE

AIM:-

To point out the various etiologies and to study the clinical profile of seizure disorder in children >2 months to <12 yrs of age.

PRIMARY OBJECTIVE:-

To know the etiology, pattern, precipitating factors, control and follow up of seizures in children between 2-12 yrs of age admitted in a tertiary.

SECONDARY OBJECTIVE:-

To follow these cases for a minimum period of 6 months to report any change needed in the current treatment protocol.

MATERIALS &METHODS

STUDY AREA

Department of Pediatrics, Govt. Theni Medical College, Theni.

STUDY POPULATION

INCLUSION CRITERIA

- All Children admitted for seizure between age 2-12 yrs.

EXCLUSION CRITERIA

- Children < 2 months or >12 yrs of age.
- Children with Simple febrile seizure disorder

STUDY DURATION

April 2014 to March 2015

Children being admitted to the pediatric department of G.T.M.C.H, Theni either in P.I.C.U or ward for the complaints of seizure disorder >2 to < 12 yrs of age, were examined after getting duly informed consent from the parents.

Past history of seizure, birth history, family history and developmental history were mainly included into the study, rather many more criteria were also examined which have been included in the proforma

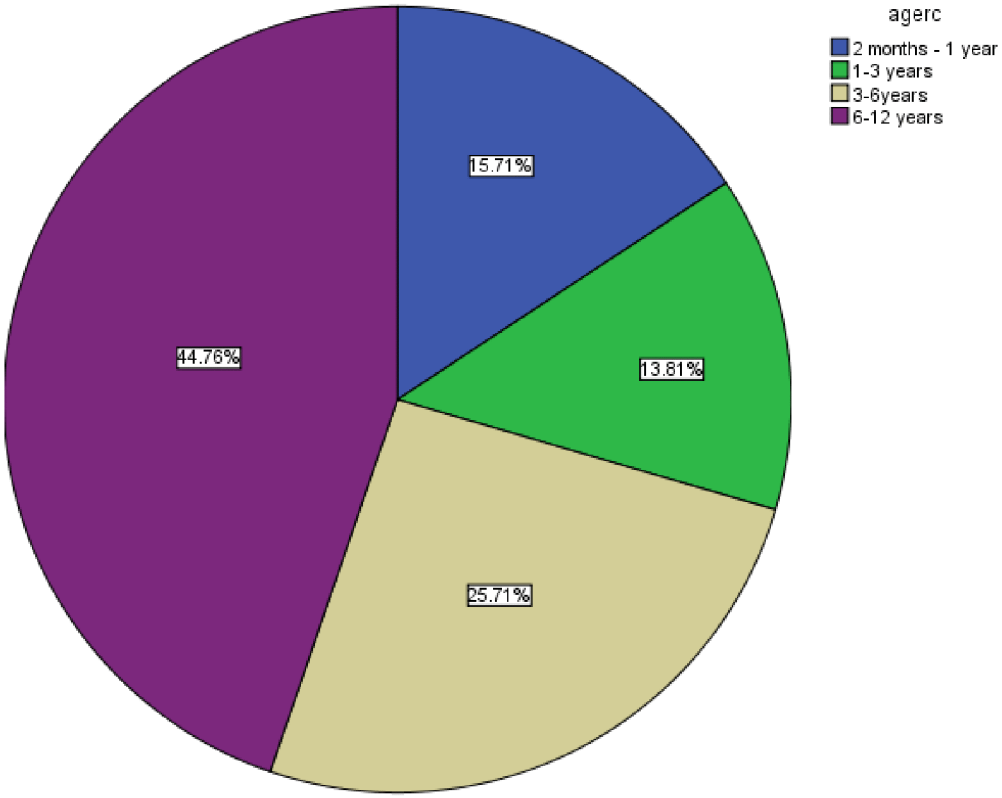
Thorough clinical examination will be done, including the neuroimaging, EEG findings and CSF examination if necessary. Etiology will be determined taking into consideration the risk factors any significant natal or ante natal history, birth history, family history of seizure, positive examination findings and examination reports.

The seizure will be classified according to the pattern of seizure and acute episodes will be treated.

The children will be follow up for 6 months after being discharged to determine the recurrence of seizure episodes during that period.

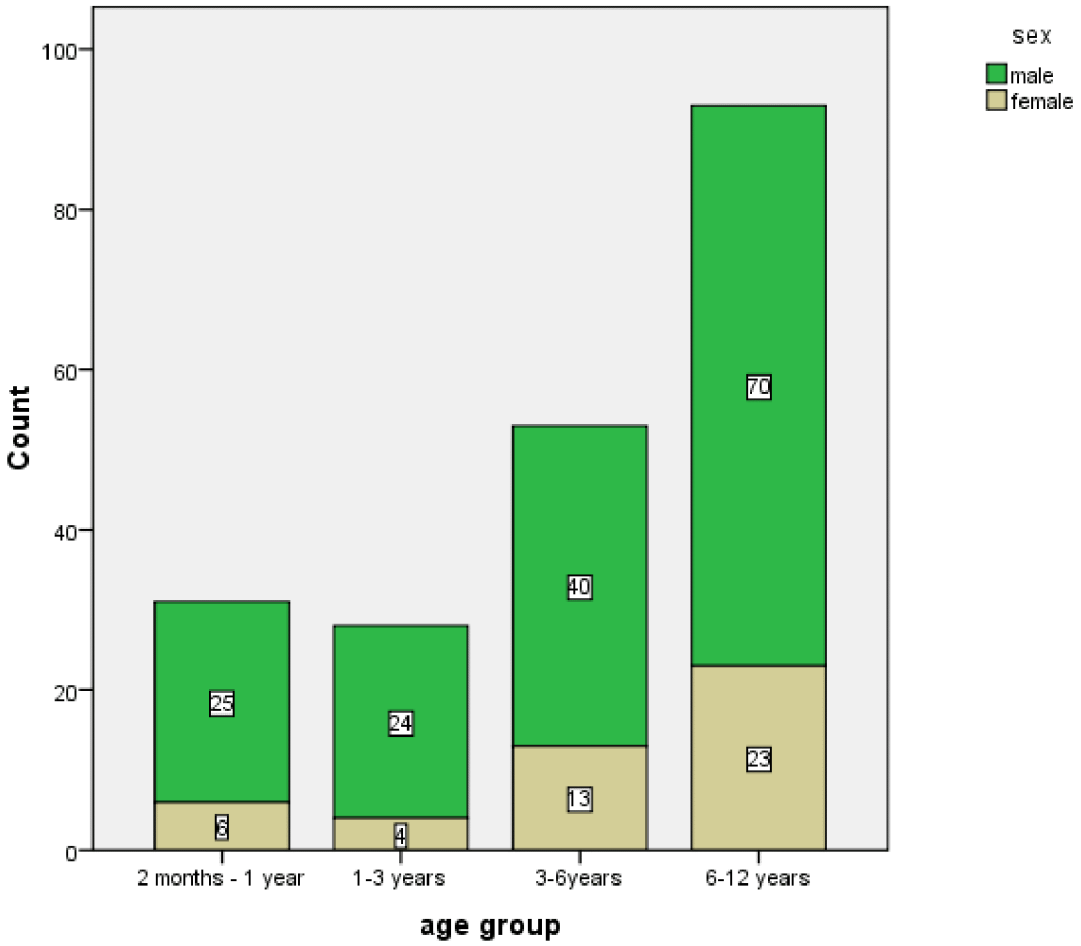
Data will be compiled in excel software and will be analyzed for etiology, topographical pattern, precipitating factors, control and their follow up.

OBSERVATION & ANALYSIS



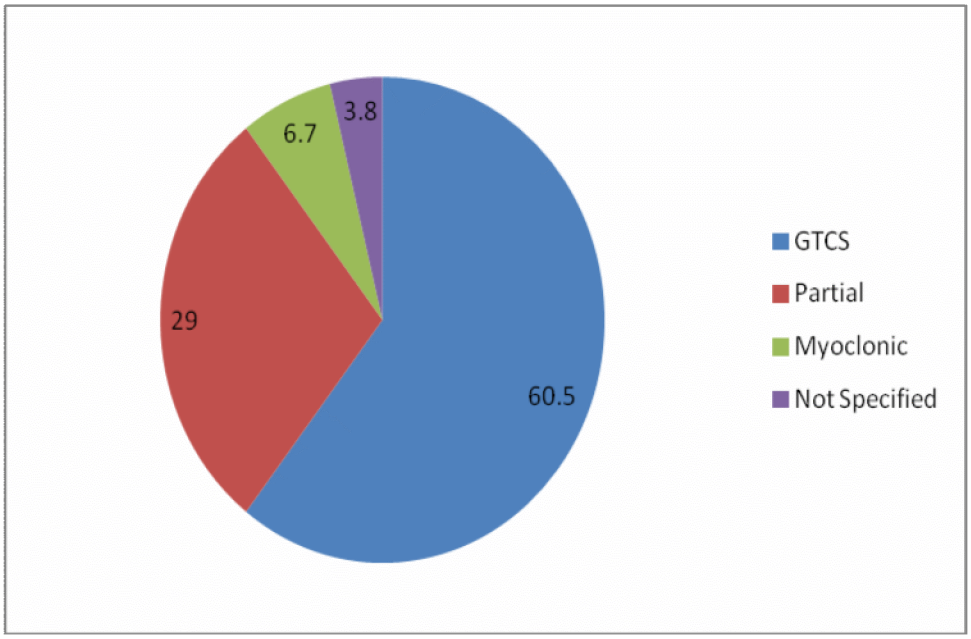
Most cases were from 6 to 12 years followed by 3-6 years then 2mths-1 year & lastly by 1-3 years, showing a topographical distribution to be more in school going children.

Figure: Age and Sex distribution in study participants



Irrespective of the age distribution males are found to be more prone to seizures as compared to the females.

Figure: Type of seizures in study participants



GTCS was found to be most dominating followed by partial & myoclonic, rest are the cases where the history was unsatisfactory

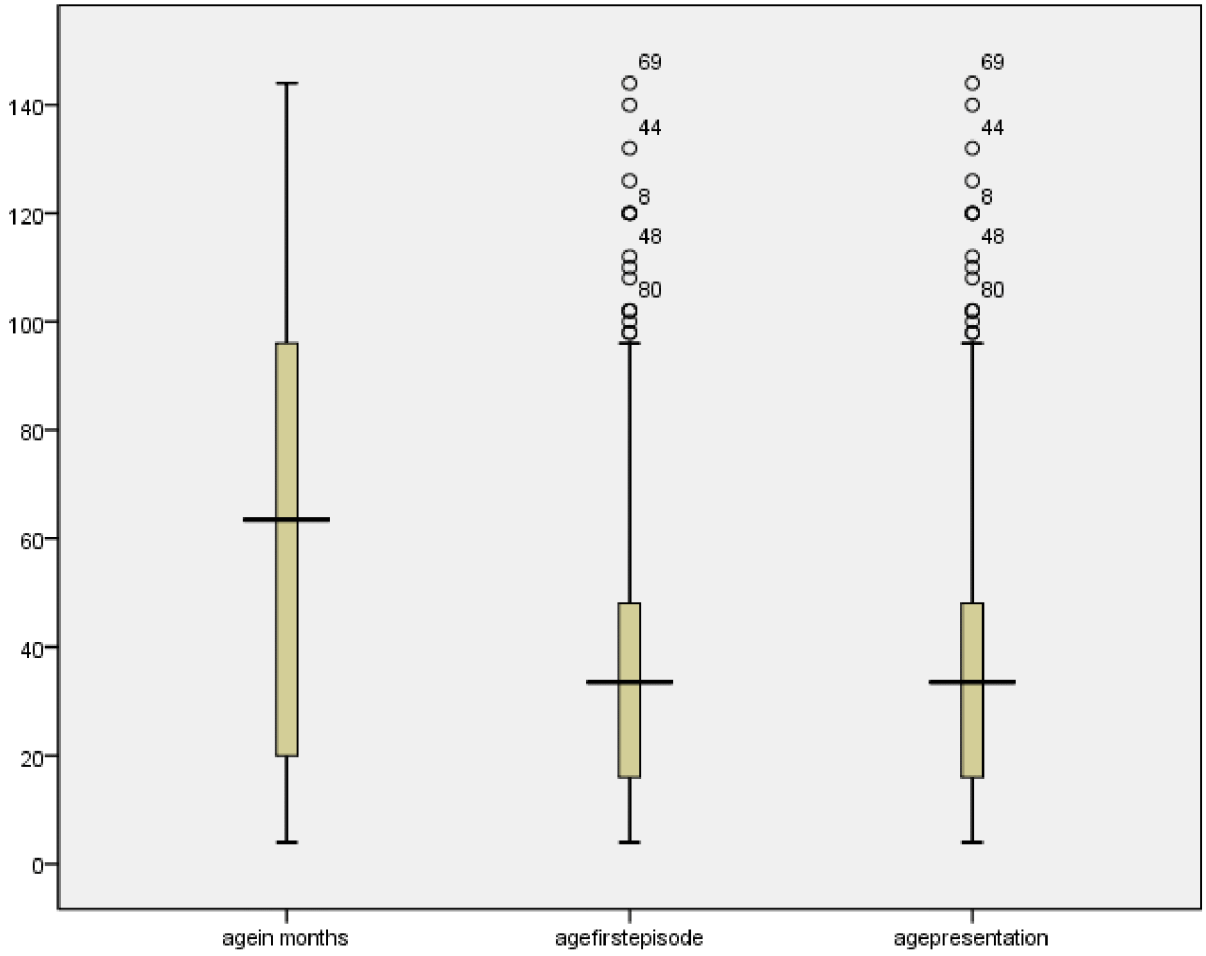


Figure: Age, age at first episode and age at presentation in study participants

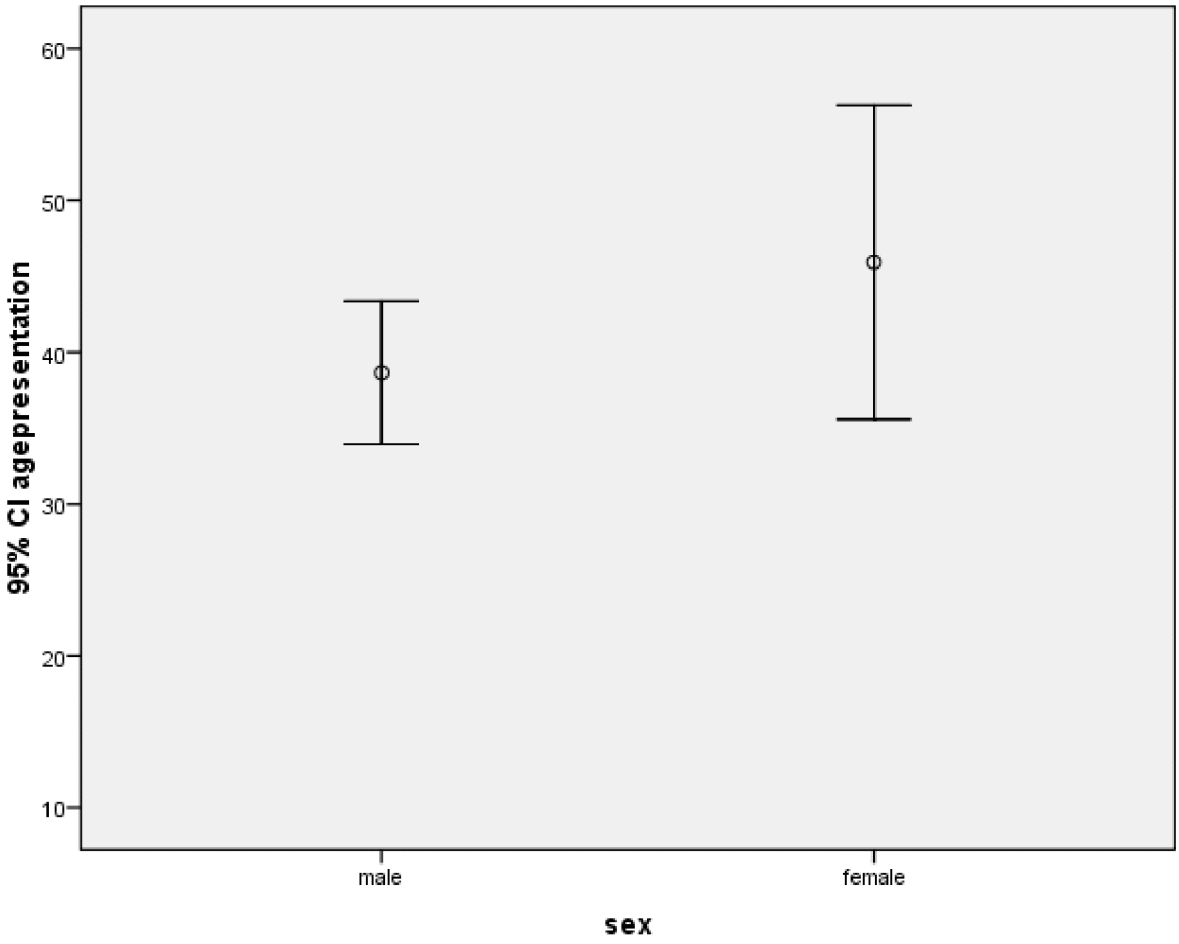


Figure: Age at presentation in males and females

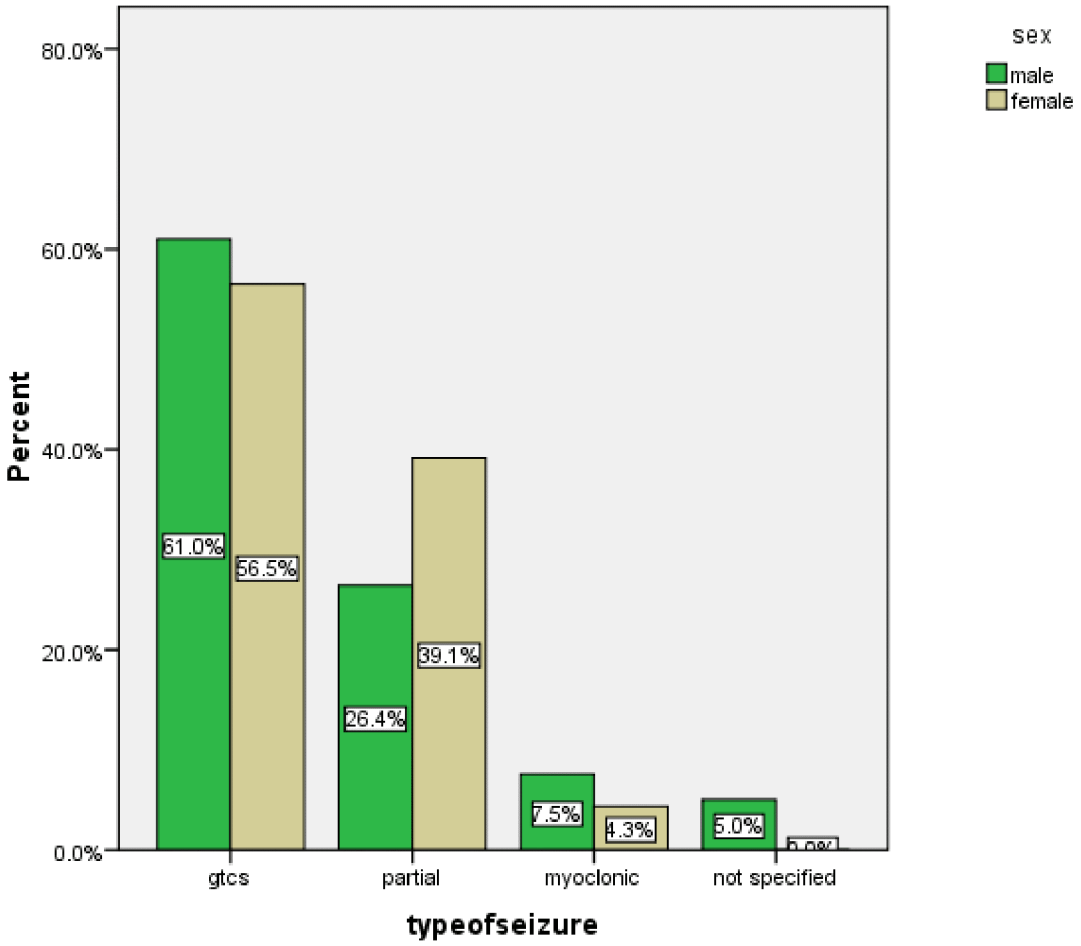


Figure: Sexwise distribution of type of seizures

		Type of seizure				Chi sq	P value
	Sex	gtcs	partial	myoclonic	not specified		
	male	97(61)	42(26.4)	12(7.5)	8(5)	6.027	0.420
	female	26(56.5)	18(39.1)	2(4.3)	0		
Total		127	61	14	8		
		60.5%	29.0%	6.7%	3.8%		

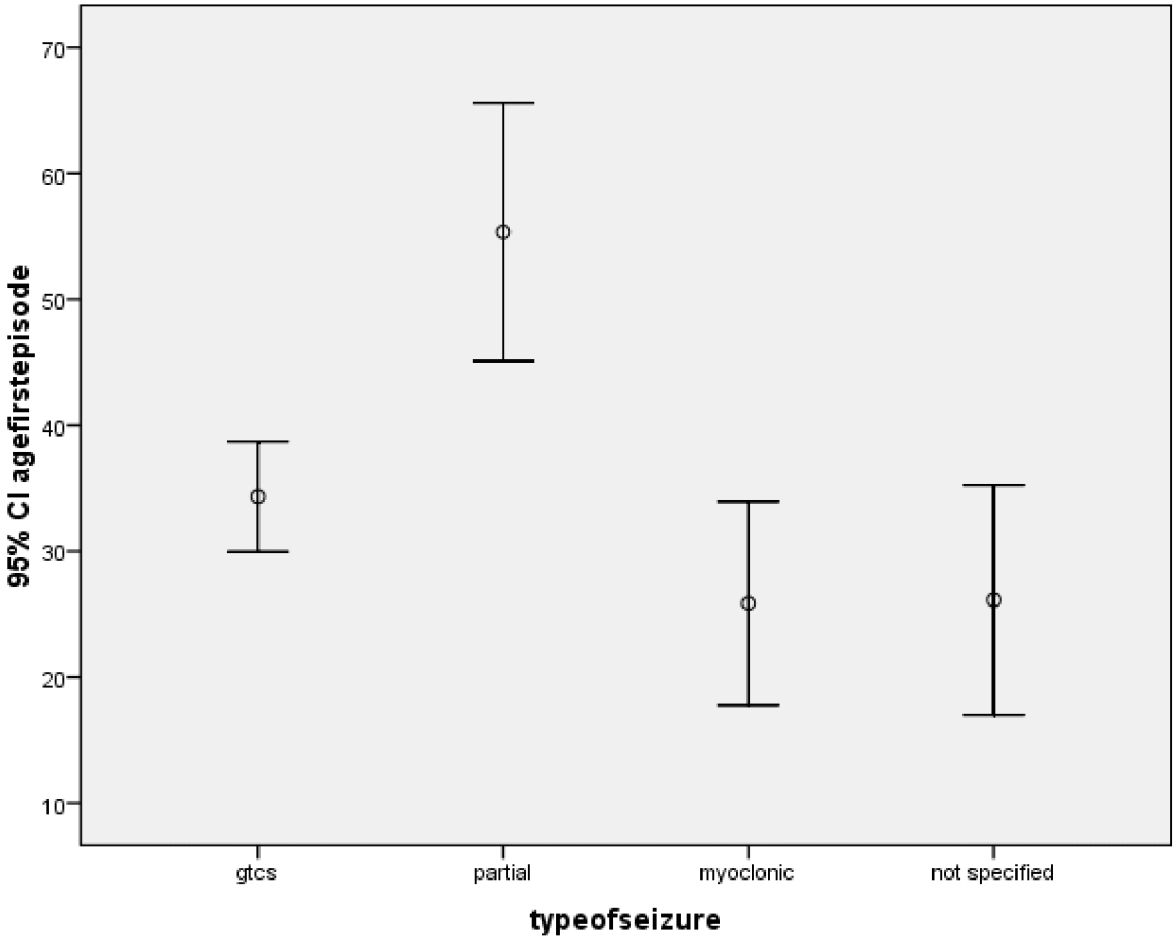


Figure : Age at first episode in different types of seizures

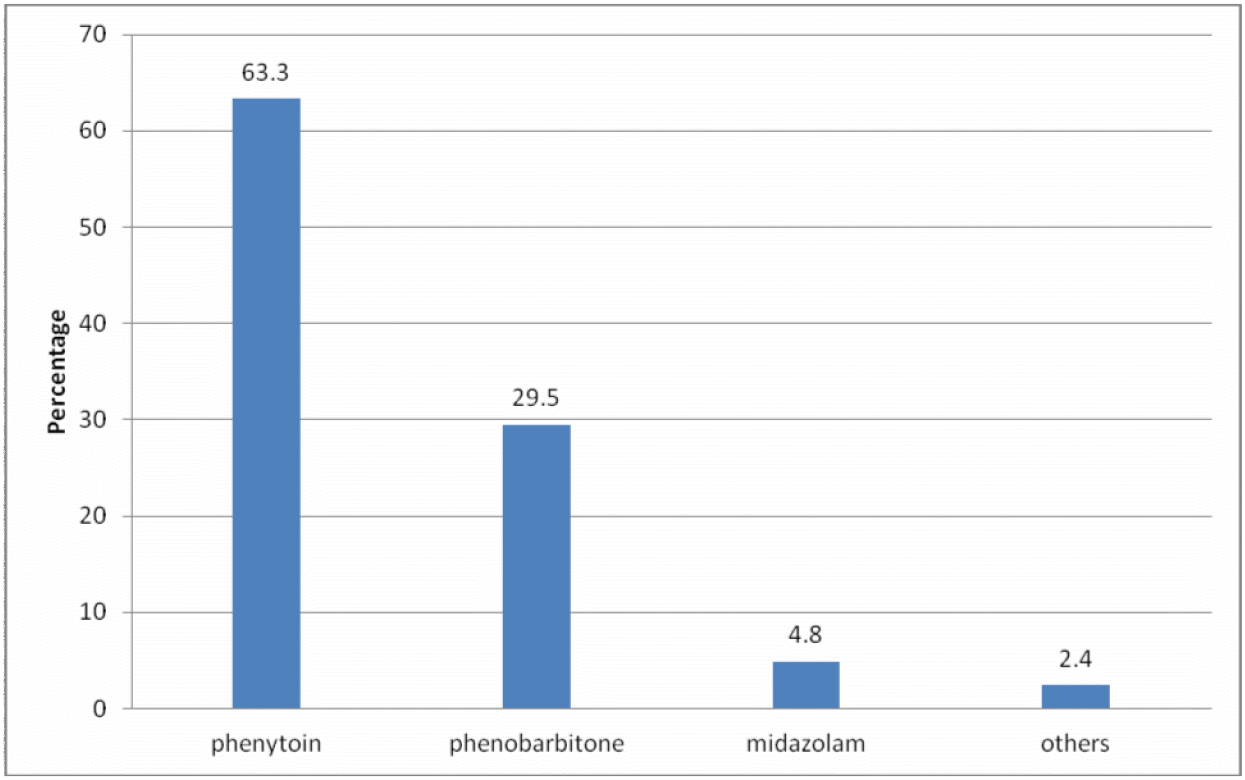


Figure: Drugs prescribed in acute episode

Most cases got controlled by single drug only, MDZ was needed in 4.8% & others included pyridoxine, leviteracetam, dextrose, calcium etc.

TABLES

PROFILE OF STUDY PARTICIPANTS

S.No	Characteristic	Number	Percentage
1.	Sex		
	Male	163	77.6
	Female	48	22.8
2.	Area		
	Rural	152	72.4
	Urban	58	27.6
3.	Education of parents		
	Literate	73	34.8
	Illiterate	137	65.2
4.	Consangunity	100	47.6
5.	Family history	37	17.6
6.	Previous treatment	96	45.7

PROFILE OF STUDY PARTICIPANTS

Variable	N	Minimum	Maximum	Mean	Std. Deviation
Age in months	210	4	144	64.64	41.984
Age first episode	210	4	144	39.57	30.972
Age presentation	210	4	144	39.71	31.106
Duration minutes	210	2	60	14.68	8.668
Frequency in a year	210	1	2	1.94	.242
Head circumference	210	35.1	55.1	46.840	5.2529

With a n value of 210, minimum age taken into consideration was 4 mths with a maximum of 12yrs with a mean of 64.64 & std devn of 41.98.

Age of 1st episode minimum was 4 mths with maximum of 12yrs, mean- 39.57 & std devn- 30.972.

Age of presentation was minimum at 4 mths with maximum of 12 yrs, mean- 39.71, std devn- 31.106.

Frequency in a year with a minimum of 1 & maximum of 2, with a std devn of 0.242

TYPE OF SEIZURE

S.No	Type of seizure	Number	Percentage
1.	Gtcs	127	60.5
2.	Partial	61	29.0
3.	Myoclonic	14	6.7
4.	Not specified	8	3.8

TOTAL OF GENERALIZED 127 CASES:-

GTC	**68**	**53.2%**
TONIC	**34**	**26.8%**
CLONIC	**10**	**7.5%**
MYOCLONIC	**5**	**4.3%**
ABSENCE	**5**	**4.1%**
ATONIC	**5**	**4.1%**

PARTIAL OF TOTAL 29 CASES:-

(1) Simple	**6**	**19%**
(2) Complex	**10**	**35.3%**
(3) Secondary generalized	**13**	**45.7%**

INVESTIGATION REPORTS IN STUDY PARTICIPANTS

S.No	Investigation finding	Number	Percentage
1.	Abnormal EEG	190	90.5
2.	Neuroimaging		
	Normal	160	76.1
	cerebral edema	32	15.2
	Tuberculoma	3	1.4
	Calcification	4	1.9
	Cerebral atrophy/HIE	11	5.2
3.	Abnormal Fundus	26	12.4

EEG was found to be most sensitive in pointing out the seizural activity. Neuro imaging was found to be normal in most of the cases, followed by cerebral oedema in case of meningitis or encephalitis.

Some cases were also sorted out to be SOL, amongst most common cause was NCC or tuberculoma. Fundus was found to be abnormal in 12.4% of cases.

ETIOLOGY OF SEIZURES

S.NO.	CAUSE	FREQUENCY	PERCENT
1.	No cause ascertained	128	60.9
2.	Birth aspx	25	11.9
3.	Camphor	5	2.3
4.	Cp/mr	33	15.7
5.	Htn	2	0.9
6	Hypocalcemia	2	0.9
7	Hypoglycemia	4	1.9
8	Hyponatremia	4	1.9
9	Ncc	4	1.9
10	Trauma	2	0.9
11	Tumor	1	0.5

No causes were found in 60.9% of cases, followed by CP/MR & birth asphyxia in 15.7 & 11.9% of cases respectively, rest other were found to be either toxic encephalopathy, metabolic cause, **HT**, SOL or other symptomatic seizures.

EFFECT OF SEIZURE

S.No	Characteristic	Number	Percentage
1.	Absenteeism at school	8	3.8
2.	NCM	12	5.7
3.	Injuries	24	11.4
4.	FND	3	1.4

Effect of seizure was also taken into consideration to know weather the seizure is affecting the day to day life of the baby including the absenteeism at school, any seizure related injuries, any FND & found out to be affecting 3.8% in case of absenteeism, some children having seizure were of lesser age & were not being admitted to the school.

Injuries were found in 11.4% & FND were found in 1.4% with NCM in 5.7%

DRUGS PRESCRIBED FOR STUDY PARTICIPANTS DURING THE ACUTE EPISODE

S.No	Drugs	Number	Percentage
1.	Phenytoin	133	63.3
2.	Phenobarbitone	62	29.5
3.	Midazolam	10	4.8
4.	Others	5	2.4

Phenytoin was found to be effective in most of the cases in controlling the acute episodes rather most of these cases reported to be 1st episodes, most of the old cases esp cp/mr needed either 2 or more drugs even MIDZ infusion was needed in some cases in 4.8% of cases.

Other included dextrose infusion, 10% calcium gluconate, pyridovine, leviteracetam etc..

DRUGS PRESCRIBED FOR STUDY PARTICIPANTS AT DISCHARGE

S.No	Drugs	Number	Percentage
1.	Valproate	147	70
2.	Phenobarbitone	32	15.2
3.	Phenobarbitone&others	1	0.5
4.	Diazepam	6	2.9
5.	Diazepam/valproate/ others	1	0.5
6.	Diazepam/valproate	3	1.5
7.	valproate/ others	1	0.5
8.	Others	1	0.5

SVP was found to be most effective leading to less recurrence & reduced readmission if prescribed in proper dosage. Dose as low as 10mg/kg was prescribed in most of the cases with regular follow up of the LFT.

Cases of cp/mr were generally needed combination of more than 2 drugs.

NEURO OPINION

S.No	Opinion	Frequency	Percent
1.	Seizure disorder	94	44.7
2.	Atypical febrile **SD**	51	24.2
3.	Symptomatic seizure	65	30.9

Compliance with treatment= 177(84.3)

SD was found to be the most common cause with 44.7%, followed by ATFBS of 24.2%, & symptomatic seizures of 30.9%. Symptomatic seizures included meningitis, encephalitis, metabolic causes or any SOL.

OUTCOME:-

	Percent	**Frequency**
Discharged normally	89.14%	189
Discharged with deficit	5.4%	11
LAMA	4.1%	8
Death	1.4%	3

Out of 210 cases admitted, 89.14% were discharged successfully, 5.4% had deficit, 4.1% were LAMA & unfortunately death occurred in 1.4%.

Most of the cases who developed deficit reported late to the hospital.

RISK FACTORS DETERMINING POOR OUTCOME

Out of varies risk factors analyzed for predicting poor out come, Age < 1 year, increasing distance from the place of onset, duration >1 hour, no proper pre hospital therapy, SaO2 of <92% at arrival, refractory shock in ER that is uncorrected even after starting ionotropes, on IPPV, refractory SE, acidosis at arrival that is low HCO3 and supported by academia in ABG, acute CNS infection as the underlying cause if SE were some of the risk factors we found. Odds ratio, 95% confident interval, P values were calculated for poor out come group comparing with good outcome group and univariate analysis was done for all these risk factors.

Of all the risk factors mentioned above, age <1 years and < 6 years (p=0.05), refractory shock (P=0.07) were not statistically significant to influence the outcome adversely. **Increasing distance (that is each Km increase in distance increases the odds ratio by 1.7), duration of > 1 hour, no proper pre hospital therapy, SaO2 < 92% at arrival, acidosis at arrival, need for IPPV, refractory SE, acute CNS infection** were significant risk factors.

Finally at the end of multiple logistic regressions of all the risk factors, only 4 factors namely increasing distance from the place of onset of seizures to GTMCH, duration of seizures, need for IPPV, acute CNS infection as the

etiology are statistically significant risk factors. They are independent risk factors influencing poor outcome.

Incidence of SE among male children is more than female children in this study and the male predominance is not statistically significant ($p>0.05$) and male, female distribution is equal in other studies also. Mean age is 3 years and 5 months observed in this study where as mean age of seizure is 2 years and 4 months noted in Mah JK et al 89 study, and Mean age of 56.6 ± 46.5 months observed in Kalra Veena et al 97 study. Most of the cases of SE in children occurred in younger age group.

Garzon E 84 observed that SE incidence peaked in the first years of life, and 56.7% cases were < 5 years in Kalra Veena 97 study. Mortality is also high in this age group. 85% of mortality occurs in the age group of < 3 years.

Outcome is determined by age, duration and underlying cause. Age < 1 year, duration of >1 hour, acute CNS infection as the underlying cause are predictors of poor outcome seen in varies studies. Young age <12 months and duration >60 minutes associated with adverse outcome concluded in Kwong et al 83 study, deaths were correlated to etiology and patient's age concluded in Garzon E 84 study, the group with Seizure lasting <30mins had a lower mortality as compared with seizure duration > or = 30mins observed in Towne AR et al 85 study. Sahin et al 87 concluded that the mortality in RSE

was related to etiology age and EEG findings and predictors of poor outcome were older age (OR = 1.04,95% CI 1.01 – 1.07), delay in treatment (OR = 9.73,95% CI 1.58 – 59.96) and CNS infection 9 OR = 30.27 95% C 3.14-292.19) seen in Hui AC et al 88 study. Outcome related to etiology, duration, and age is a minor factor

Observed in Dunn DW 90 study, mean seizure duration was 1.5 ± 2.8 hours in those children with a normal outcome, 1.7 ± 1.2 hours in those survivors with an abnormal neurological outcome ($P > 0.05$), and 6.8 ± 12 hours in those who died ($P < 0.05$) and both the duration and etiology of status epilepticus affect the outcome concluded by Simon J et al 92.1.

In our study also duration > 30mins, increasing distance from the place of seizure onset, acute CNS infection, need for IPPV were significant independent risk factors that predict poor out come.

Commonest seizure type is GTCS. Prolonged seizure in many cases resulted in SE due to neuroelectromechanical dissociation.

Proper pre hospital therapy is associated with good out come observed in this study. No or improper pre hospital therapy is a significant risk factor for poor outcome in univariate analysis.

Kwong et al 83 concluded that Pre hospital Rx with BZD reduces adverse outcome.

Some of them were apneic at arrival and 100% needed supplementary O2 either BVM with 100% O2 or O2 through non rebreathing mask in this study. O2 through non rebreathing mask can be given only when the respiration is regular and adequate only in case of seizures<30 mins because all CSE cases and most of the NCSE cases the respiratory muscles also involved in seizure activity resulted in apnea. Apnea is not the contra indication for giving AEDs but is an indication for initiating BVM in them. Most of them were presented with shock also and needed fluid boluses and ionotropes support . In hemodynamically unstable patients, phenytoin should be used cautiously or it can be substituted with other AEDs. FP is found to be safe in these patients but it was not used as an AED in this study. Commonest side effect observed after stating phenytoin infusion was shock and hypotension rarely arrhythmias needed ionotropes support.

Frequent monitoring of BP and HR/rhythm perfusion status is must. Preferably phenytoin is avoided in young infants of <3 months. 2 cases were found to have hypotension at arrival and subsequently died.

Horn drop: Study observed that symptomatic seizure and refractory SE and associated with poor outcome than idiopathic or febrile seizures and young age < 12 months duration of seizure > 60 minutes are associated with adverse outcome.

Causes of SE : Idiopathic -30%., fever- 25%, acute symptomatic- 35%, remote symptomatic- 15%, progressive- 5%.

Most common causes are AED withdrawal or non compliance, metabolic disturbance, drug toxicity, CNS infection, CNS tumor, Refractory epilepsy, head trauma, febrile SE.

CNS infection is more common in our setup but it is low in western countries (5%) due to the implementation of Hib, Pneumococal vaccination and improvement in quality of life style, environmental sanitation and safe water supply. We come across only 2 new neurological sequele during this study. Both were due to acute CNS infection. 18/114 new neurological deficit observed in Dunn W 91 study and 17/193 new neurological deficits occurred in Maytal J et al 98 study.

Higher mortality in this study is mainly due to the underlying cause than seizure itself. Most of the cases of SE were young children of <6 years of age and morbidity is also high in young children of <3 years who had 85% mortality.

There is no significant sex difference.

Commonest seizure type is GTCS.

Most of them were requiring supplementary oxygen at arrival and most of them were apneic, hypoxic and in shock.

Some of the children had hypoglycemia and 11% & all of them received 25% dextrose.

Common causes of Seizure are acute CNS infection, septic shock, idiopathic epilepsy, febrile SE and CNS co morbidity like CP.

Febrile seizure and idiopathic epilepsy were associated with good prognosis. All the children in FS group recovered completely without any sequele. CNS infection and septic shock were associated with poor outcome. New neurological sequele occurred in 2 cases both of them had acute CNS infection as the underlying etiology. Long term outcome in these survivors need to be evaluated further.

Acute CNS infection, duration of SE, distance travelled to seek medical advice and respiratory failure requiring IPPV, are independent risk factors that influence the outcome adversely.

Refractory seizures or SE is associated with poor outcome and prolonged hospital stay. Most of them responded to midazolam. All cases of RSE were intubated using midazolam IV as the sedative and neuromuscular blockade avoided. Common complication of midazolam infusion is shock and noted only with higher doses >6 mcg/kg/min and managed with ionotropes.

1. Common cause of SE in our part of the country is acute CNS infection and this results in higher mortality, morbidity and later neurological sequele.

Acute CNS infection is one of the independent risk factor for poor outcome in SE. Vaccinating with Hib, Pneumococcal vaccines and prompt use of antimicrobial therapy in suspected cases to be undertaken to prevent acute CNS infection.

2. Duration and distance travelled to seek medical advice are also important risk factors influenzing poor outcome. So, early institution of proper time framed therapy even by the nearest hospital will improve the outcome. At least, proper pre hospital therapy like the use of IM/PR/IV Midazolam or IV/PR diazepam and taking care of airway and breathing with supplementary O2 while transporting the child from the peripheries could result in better outcome in SE children.

3. Many of the complications and consequences can be managed successfully with anticipation and early intervention like shock, respiratory failure, aspiration pneumonias, hypo/hyper glycemias, dyselectrolytemias.

4. A bed side EEG will be useful in managing NCSE as the electrical SE or neuro EMD are also associated with same neuronal damage and mortality as CSE. Even if EEG is not available a high index of clinical suspicion of NCSE should be there to identify ongoing seizures in partially treated comatose children. The clinical signs such as persistent apnea, unresponsiveness, defective DEM, nystagmus, conjugate deviation, excessive

secretions and disproportionate tachycardia are some of the very important clues in identifying NCSE.

5. Management of vital signs and underlying cause of seizure along with specific AED therapy are the priorities in the management of seizures.

6. Midazolam is safe and effective for the treatment of SE in children followed by intubation & respiratory support.

Data on outcome in seizure disorder in children is sparse in India. Seizures are one of the most common emergencies, we managed in our hospital and we see the large number of seizure in children, in our part of the country. As a tertiary level referral hospital, we managed those cases that were referred as refractory seizures. Many children come from long distances with prolonged seizures. They do not have effective pre hospital therapy, proper referral and transport services.

Though we managed many seizures successfully, we do come across poor outcome in seizures. If the risk factors influenzing poor out come are identified, some of the factors can be modified and the high risk groups, who are going to have poor outcome can be managed properly.

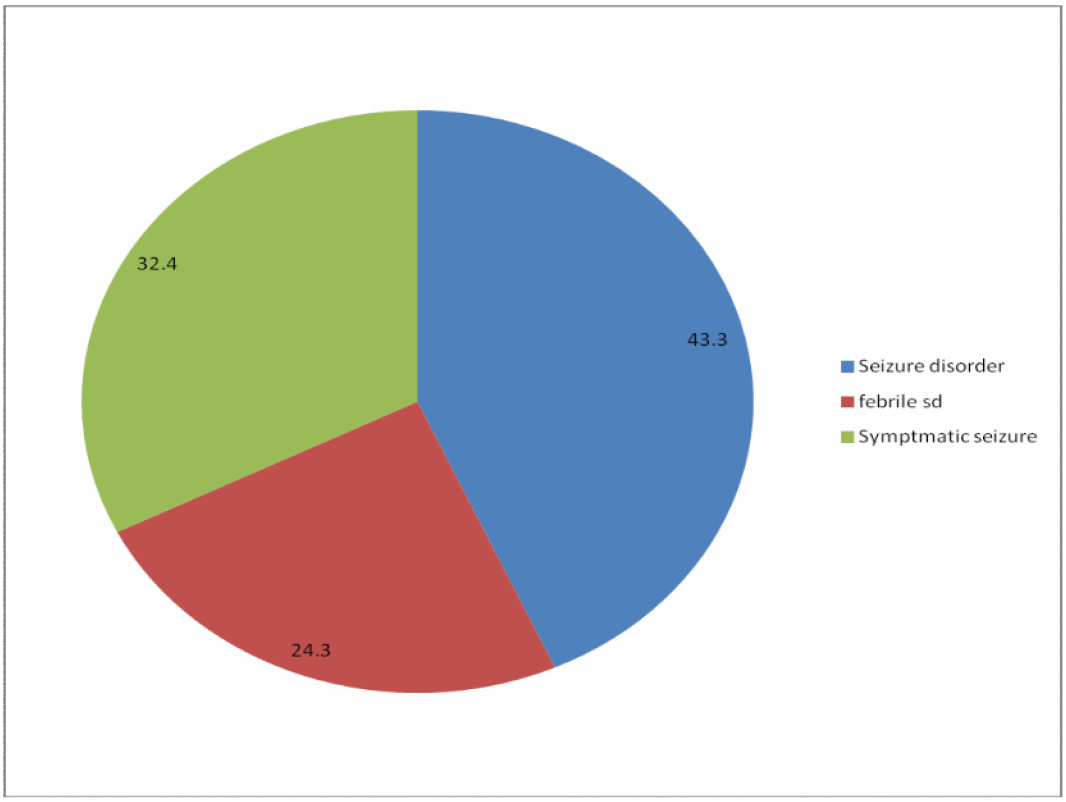


Figure: Etiology of seizure- Neurologist opinion

SD came out to be the most common cause, followed by ATFSD & symptomatic seizure.

DISCUSSION

In this study conducted during a period of 1 year a total of 210 cases were admitted and followed. 75.7% were males and 21.9% were females, out of which 72.4% were from rural area & 27.6 from urban, 34.8% were literate but 65.2% illiterate, 47.6% were consangious high is a very high rate. Family history was present in 17.6% & previous treatment was present in 45.7% of cases.

Most of the aspects were compared during this study including age of 1st episode , age of presentation, types of seizures and frequency in a year that can affect the life and profile of the patient.

In cases of children going to school absenteeism in school was taken into consideration, to know weather the seizure affects the school life and daily activities of the child. It was found that it accounts for total 3.8% of cases, neurocutaneous markers were found in 5.7% of cases, seizure related injuries was found in 11.4% of cases and focal neurological deficit was found in 1.4% of cases.

Considering the type of seizure, GTCS was found to be the commonest 60.5%, followed by partial seizure in 29%, myoclonic in 6.7% & non specified in which the history as not confirmed in the remaining 3.8% of cases.

Abnormal EEG was found in 90.5% of cases, and found to be most sensitive in determining the seizural activity of the child.

Neuroimaging was done in all cases and if required CT/MRI/MRA/MR SPECTROSCOPY was also done to determine the cause.

It was divided into either normal or abnormal findings. Normal was found in 76.1% of cases.

Abnormal findings included cerebral oedema 15.2%, tuberculoma in 1.4%, calcification in 1.9%, and cerebral atrophy in 5.2% of cases.

Fundus examination was done in all the cases and was found to be abnormal in 12.4% of the cases.

Not only the neuroimaging and fundus but the clinical presentation, history and every aspect was taken into consideration for deriving a suitable diagnosis.

No causes were ascertained in 60.9% of cases & included into asymptomatic seizure, symptomatic seizures included:-

(a) Camphor poisioning- 2.3%

(b) Htn encephalopathy- 0.9%

(c) Hypocalcemia - 0.9%

(d) Hypoglycemia - 1.9%

(e) Hyponatremia - 1.9%

(f) Ncc - 1.9%

(g) Trauma - 0.9%

(h) Tumor -0.9%

Birth asphyxia was found as a cause in 11.9% of cases with CP/MR in 15.7% of cases.

For treating the seizure universal treatment protocol was followed & was found that phenytoin was effective in 63.3%, PB in 29.5%, MDZ was needed in 4.8%, others like calcium infusion, dextrose infusion or correction of HTN or hyponatremia in 2.4% of cases.

The patients were discharged and followed up regularly according to repeat EEG & clinical condition.

SVP was found to be effective in most cases including 70%, PB in 15.2%, DZP in 2.9% & rest were discharged with 2 or more drugs.

CONCLUSION

1. SD was found to be the most common cause in children from 2mths to 12 yrs.
2. Atypical febrile SD was the 2nd most common cause.
3. Symptomatic seizure was found in rest cases.
4. Symptomatic seizure included meningitis, encephalitis, metabolic & electrolyte abnormalities, HTN & SOL.
5. Amongst SOL tuberculoma, NCC, Pineal gland tumor was found to be common.
6. Most of the population were from rural areas and were not having good transport facilities, as the prolongation of the seizure will led to SE and more worse outcome.
7. Only 34.8% were educated compared to rest, and were more concerned about the diseased state of the child.
8. NCM was found only in 52.4% cases showing a very great incidence of consanginious marriage in this region.
9. Family history of seizure was also found to be significant.
10. Fundus examination gives lots of clues including disc oedema, blurring of disc margins & should be done in all cases
11. Diagnosis should be done after stabilizing the patient's general condition.

12. Detailed history, examination and investigations should be taken into consideration before arriving to a diagnosis.

13. GTCS was found to be most common but any seizure in infants, 2mths to 18 mths, focal, associated with fever should be treated vigorously including CSF examination.

14. SVP showed to be most promising as low as 10mg/kg.

15 . PB should be replaced by SVP in all children more than 1yrs of age with a regular follow up of LFT.

16. Lastly patients should be followed up regularly to watch for improvement or detoriation & should be intervened quickly.

LIMITATIONS

Simple febrile seizure.

Seizure <2 months & >12 years.

Low socio economic group were not able to do some investigations

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PROFORMA

NAME: **AGE & SEX:** **IPO No.-**

ADDRESS:-RURAL/URBAN

TRANSPORT FACILITY:

AGE OF ONSET OF FIRST EPISODE:

AGE OF PRESENTATION:

LEVEL OF EDUCATION OF PARENTS/ CAREGIVERS:

PARENTAL CONSANGUNITY:

FAMILY H/O EPILEPSY:

PREVIOUS TREATMENT HISTORY:

TYPE OF SEIZURE:

GTCS:

PARTIAL:

MYOCLONIC:

NOT DEFINED:

DURATION OF SEIZURE:

SCHOOL PERFORMANCE: (in terms of absentism)

FREQUENCY OF SEIZURES:

EXAMINATION FINDINGS:

Neuro cutaneous markers

Head circumference

Seizure related injuries

Focal neurological deficit

Other findings

NEURO IMAGING:

EEG FINDINGS:

FUNDUS EXAMINATION:

C.S.F Examination (if necessary):

OTHER EXAMINATION(if significant):

ETIOLOGY:

BIRTH ASPHYXIA:

C.N.S Infection/Encephalitis:

TRAUMA:

TOXINS:

IDIOPATHIC: **OTHERS:**

CO-MORBIDITY:

TREATMENT HISTORY:

ACUTE EPISODE:

DISCHARGED DRUGS:

COMPLIANCE:

FOLLOW UP:

NEUROLOGIST'S OPINION:

KEY TO MASTER CHART

address
- rural-1
- urban-2

transport
- good-1
- bad-2

edun parents
- educated-1
- uneducated-2

consanguinity
- consanginious-1
- nonconsanginious-2

famhist
- yes-1
- no-2

prev treatment
- yes-1
- no-2

types of seizure
- gtcs-1
- focal-2
- myoclonic-3
- others-4

frequency
- frequent-1
- notfrequent-2

neurocutaneous markers
- yes-1
- no-2

injuries
- yes-1
- no-2

fnd
- yes-1
- no-2

imaging
- normal-1
- abnormal-2

eeg
- normal-1
- abnormal-2

fundus
- normal-1
- abnormal-2

compliance
- good-1
- bad-2

acute drugs
- phenytoin-1
- phenobarbitone-2
- midazolam-3
- others-4

discharge drugs
- phenytoin-1
- phenobarbitone-2
- diazepam-3
- sod valproate-4
- others-5

S.No.	ipno	agein months	sex	address	transport	agefirstepisode	agepresentation	eduparents	consanguinity	famhistory	prevtreatment	typeofseizure	durationmins	absenteeism	frequencyinayear	ncm	hc	injuries	fnd	others	neuroimaging	eeg	fundus	csfglucose	csfprotein	csfcells	csfculture	etiology	comorbidity	compliance		neuroopinio		acute drugs	discharge drugs
1	12235	42	f	1	2	42	42	1	2	2	2	1	5	1	2	2	46.9	2	2		1	1	1					camphor		1		toxic encephalopathy		2	4
2	11985	60	f	1	2	36	36	1	2	2	1	1	10	2	1	2	50.8	2	1		2	2	1							1		seizure disorder		1	4
3	11823	42	f	1	2	11	11	1	2	2	1	1	20	2	2	2	48.5	2	2		1	2	1						lbw	1		seizure disorder		1	4
4	11251	72	m	1	1	72	72	1	1	1	2	1	10	1	2	2	50.1	2	2		1	2	1							1		seizure disorder		1	4
5	42694	48	m	2	1	48	48	1	1	1	2	1	2	2	1	2	49.2	2	2		1	2	1							1		seizure disorder		1	4
6	12792	72	m	1	2	18	18	1	2	2	2	1	10	2	2	2	48.1	2	2		1	2	1							1		seizure disorder		1	4
7	18764	60	m	1	2	24	24	2	2	1	2	1	5	2	1	2	42.5	2	2		4	2	1							2		cp/mr/seizure disorder		2	4&5
8	9723	120	m	1	2	120	120	2	1	1	2	1	10	2	1	2	52.4	2	2		1	2	1					birth aspx		1		seizure disorder		1	4
9	8069	18	m	1	2	18	18	2	1	2	2	1	60	1	1	2	45.4	2	2		1	2	1	82	47	200		cns infn		2		encephalitis		1	2
10	37117	54	f	1	2	54	54	1	2	2	2	2	10	2	2	1	45.2	2	2		2	2	1							2		seizure disorder		1	4
11	10617	33	m	2	1	33	33	2	2	1	2	1	20	1	2	2	49.5	2	2		1	2	1							1		sd		1	3
12	9091	12	f	1	2	12	12	1	2	2	1	1	20	0	2	2	45.9	2	2		1	2	1	56	40	98				1		bacterial meningitis		1	2
13	10114	84	m	1	2	54	84	1	2	1	2	1	3	1	2	2	54.4	2	2		1	2	1							1		sd		1	3
14	10409	60	m	1	2	60	60	2	1	2	2	1	20	0	2	1	48.2	2	1		2	2	1					birth aspx		1		seizure disorder		1	3
15	10172	48	f	1	2	15	15	1	2	2	1	1	5	0	1	1	42.9	2	2		3	2	1						lbw/ micrcephly	1		seizure disorder		1	3
16	10364	36	f	1	2	36	36	1	1	2	2	1	15	0	2	2	45.2	2	2		1	2	1							1		sd		1	3
17	9105	18	m	1	2	12	12	1	2	2	2	1	20	0	2	1	45.5	2	2		1	2	1							2		febrile sd		1	2
18	9436	42	m	1	2	18	18	2	1	2	1	1	30	2	1	2	45.1	2	2		1	2	1							2		febrile sd		1	3&4
19	9640	60	m	2	2	60	60	2	2	2	2	1	5	2	1	2	43.8	2	2		2	2	1							2		febrile sd		1	4
20	11235	4	m	1	2	4	4	2	1	1	2	1	10	0	2	2	37.2	2	2		1	2	1	32	150	1150				1		bacterial meningitis		1	2

21	1234	6	m	1	2	6	6	1	2	2	2	3	15	0	2	2	38.4	2	2		1	1	1	74	34	4		hypoglycemia		1		hypoglycemic		4	
22	1287	5	m	1	2	5	5	2	1	1	2	3	10	0	2	2	36.2	2	2		1	2	1	68	32	2		hypocalcemia		1		hypocalcemic seizure		4	2&5
23	1395	8	m	1	1	8	8	2	1	2	2	2	15	0	2	2	38.1	2	2		1	2	1	39	110	115		cns infn		1		bacterial meningitis		1	2
24	1345	9	m	1	1	9	9	2	1	2	2	1	10	0	2	2	40.1	2	2		1	2	1	36	104	1123		cns infn		1		bacterial meningitis		1	2
25	1256	60	m	1	2	60	60	2	2	2	2	1	25	1	2	2	49.2	2	2		2	2	2	33	124	1129		cns infn		1		bacterial meningitis		1	4
26	1543	36	m	1	2	12	12	1	2	2	1	1	5	0	2	2	42.1	2	2		1	2	1							1		seizure disorder		1	4
27	1643	12	m	1	2	12	12	2	1	2	2	2	15	0	2	2	37.1	2	2		1	2	1	34	155	1150		cns infn		1		bacterial meningitis		1	2
28	1267	6	m	2	1	6	6	1	2	2	2	1	10	0	2	2	39.9	2	2		1	2	1	68	40	3				1		febrile sd		1	2
29	1290	48	m	1	2	12	12	2	2	1	1	1	15	1	2	2	46.4	2	2		1	2	1							1		febrile sd		1	4
30	1543	8	m	2	1	8	8	1	2	2	2	1	20	0	2	2	38.5	2	2		2	2	1	44	98	1348		cns infn		1		bacterial meningitis		1	2
31	1240	32	m	2	2	32	32	2	1	2	2	2	45	1	2	2	40.1	2	2		2	2	2	72	244	550		cns infn		1		encephalitis		3	3
32	12678	48	m	1	2	35	35	2	2	2	1	1	10	0	2	2	41.1	2	2		1	2	1							1		febrile sd		1	4
33	12789	92	m	1	2	36	36	2	1	2	1	1	15	2	2	2	44.2	2	2		1	2	1							1		sd		1	4
34	12567	74	m	1	2	44	44	2	1	2	1	1	10		2	2	46.2	2	2		1	2	1							2		sd		1	4
35	12456	4	m	1	2	4	4	2	1	2	2	2	10		2	2	38.2	2	2		1	2	1	37	154	245		cns infn		1		bacterial meningitis		1	2
36	12450	19	m	2	1	10	10	1	2	2	1	2	15		2	2	39.2	2	2		1	2	1							1		febrile sd		1	2
37	12546	96	f	1	2	96	96	2	1	2	2	2	20		2	2	48.5	2	2		2	2	1					ncc		1		ncc		1	4
38	12876	84	m	1	2	84	84	2	1	2	2	2	15		2	2	48.2	2	2		2	2	1					ncc		2		ncc		1	4
39	11345	140	m	2	2	140	140	2	1	2	2	2	10		2	2	50.1	2	2		2	2	1					ncc		2		ncc		1	4
40	11098	74	m	2	2	74	74	2	2	2	2	2	20		2	2	49.5	2	2		2	2	1					ncc		1		ncc		1	4
41	1324	62	m	1	2	62	62	2	1	2	2	2	7		2	2	45.4	2	2		2	2	2	32	130	88		tubercular		1		tbm		1	4
42	15436	74	m	1	2	74	74	2	2	1	2	2	20		2	2	42.1	2	2		1	2	2	44	128	110		tubercular		1		tbm		1	4
43	1357	60	m	1	2	60	60	2	1	2	2	2	10		2	2	44.5	2	2		1	2	2	43	154	120		tubercular		1		tbm		1	4
44	12463	132	f	1	2	132	132	1	1	2	2	2	10		2	2	46.7	2	2		1	2	2	45	148	358		tubercular		1		tbm		1	4
45	11120	96	m	2	1	96	96	1	1	1	2	2	15		2	2	48.5	2	2		2	1	1	46	134	342		tubercular		1		tbm		1	4
46	11234	78	m	1	2	78	78	1	1	1	2	2	12		2	2	46.2	2	2		1	1	1	44	162	468		tubercular		2		tbm		1	4

47	1346	9	m	1	2	9	9	2	1	2	2	1	15		2	2	38.2	2	2		1	2	1	45	254	112		cns infn		1		bacterial meningitis		1	2
48	11224	112	f	2	1	112	112	2	2	2	2	2	5		2	2	46.2	2	2		1	2	2	56	144	250		tubercular		2		tbm		1	4
49	11251	84	m	2	1	84	84	2	1	2	2	2	10		2	2	44.1	2	2		2	1	1	68	138	550		tubercular		1		tbm		1	4
50	11288	102	f	1	2	102	102	2	1	2	2	2	10		2	2	43.4	2	2		1	1	1	40	140	342		tubercular		1		tbm		1	4

51	11648	12	m	2	1	12	12	1	2	2	2	1	15		2	2	38.4	2	2		1	2	1	66	34	5				1		febrile sd		1	2
52	10641	93	m	1	2	93	93	2	2	2	2	2	20		2	2	46.4	2	2		1	2	2	42	110	350		tubercular		1		tbm		1	4
53	1267	120	m	2	1	120	120	2	2	2	2	2	10		2	2	44.1	2	2		2	2	2	40	124	134		cns infn		1		bacterial meningitis		1	4
54	12408	12	m	1	2	12	12	1	1	2	1	2	15		2	2	38.5	2	2		2	2	2	34	122	145		cns infn		1		bacterial meningitis		1	2
55	10976	8	f	1	2	8	8	1	1	2	2	2	20		2	1	36.4	2	2		1	2	2	38	176	236		cns infn		1		bacterial meningitis		1	2
56	1357	9	f	1	2	9	9	1	1	2	2	2	10		2	2	38.5	2	2		2	2	2	24	164	365		cns infn		2		bacterial meningitis		1	2
57	134689	44	m	1	2	44	44	2	1	2	1	2	8		2	2	40.1	2	2		2	2	2	35	134	654		cns infn		2		bacterial meningitis		1	4
58	13465	10	f	1	2	10	10	2	2	2	2	2	6		2	1	39.9	2	2		1	1	2	24	184	124		cns infn		1		bacterial meningitis		1	2
59	12575	11	m	1	2	11	11	2	2	1	2	1	5		2	2	38.6	2	2		2	2	2	26	153	345		cns infn		1		bacterial meningitis		1	2
60	123467	14	m	1	2	14	14	1	2	2	2	1	15		2	2	40.2	2			2	2	2	22	144	368		cns infn		1		bacterial meningitis		1	2
61	12674	15	m	2	1	15	15	1	1	2	2	1	20		2	2	40.1	1	2		2	2	2	32	176	236		cns infn		1		bacterial meningitis		1	4
62	13478	18	f	2	1	18	18	1	1	2	2	2	25		2	2	39.8	1	2		2	2	2	36	165	578		cns infn		1		bacterial meningitis		1	4
63	123478	18	m	2	1	18	18	2	2	1	2	2	17		2	2	42.1	2	2		2	2	2	28	145	322		cns infn		1		bacterial meningitis		1	4
64	12476	16	m	1	2	16	16	2	2	2	2	2	12		2	2	38.4	2	2		2	2	2	38	134	132		cns infn		1		bacterial meningitis		1	4
65	12695	36	m	1	2	36	36	2	2	2	2	2	10		2	2	43.4	2	2		2	2	2	37	100	144		cns infn		1		bacterial meningitis		1	4
66	1578	14	f	1	2	14	14	2	2	2	2	2	15		2	2	36.6	2	2		1	2	2	34	112	178		cns infn		2		bacterial meningitis		1	2
67	15604	86	m	1	2	86	86	1	1	2	2	1	20		2	2	42.9	2	2		1	2	2	86	78	490		cns infn		1		encephalitis		1	4
68	1679	96	m	1	2	96	96	2	1	2	2	2	45		2	2	44.6	2	2		1	2	1	84	84	386		cns infn		1		encephalitis		1	4

69	108	144	f	1	2	144	144	2	1	2	2	2	25		2	2	43.7	2	2		2	2	2	98	98	654		cns infn		1		encephalitis		1	4
70	18643	98	m	1	2	98	98	1	2	2	2	2	10		2	2	43.1	2	2		2	2	2	90	134	534		cns infn		1		encephalitis		1	4
71	15680	102	m	1	2	102	102	2	2	2	2	1	40		2	2	42.2	2	2		2	2	1	89	68	763		cns infn		1		encephalitis		1	4
72	10874	84	f	1	2	84	84	1	2	2	2	1	30		2	2	44.1	2	2		2	2	2	80	100	884		cns infn		1		encephalitis		1	4
73	17659	36	m	1	2	36	36	2	1	2	2	2	5		2	2	45.9	2	2		1	1	1					camphor poision		2		toxic encephalopathy		1	4
74	16578	48	m	2	1	48	48	1	1	2	2	2	15		2	2	46.7	2	2		1	1	1					camphor poision		1		toxic encephalopathy		1	4
75	19754	50	m	1	2	50	50	2	2	2	2	1	20		2	2	48.4	2	2		1	1	1					camphor poision		1		toxic encephalopathy		1	4

76	13468	46	f	1	2	46	46	2	1	2	2	2	18		2	2	44.5	2	2		1	1	1					camphor poision		2		toxic encephalopathy		1	4
77	14578	108	m	1	2	108	108	1	1	2	2	1	20		1	2	44.6	2	2		1	2	1					htn		1		htn encephalopathy		1	4
78	14678	120	f	1	2	120	120	2	1	2	2	1	15		2	2	48.9	2	2		1	2	1					htn		1		htn encephalopathy		2	4
79	14321	98	m	1	2	98	98	2	2	2	2	1	5		2	2	43.2	1	2		2	2	1					trauma		2		post traumatic encephalopathy		2	4
80	12567	102	m	1	2	102	102	2	1	2	2	2	10		2	2	40.1	1	2		2	1	1					trauma		2		post traumatic encephalopathy		1	4
81	13586	4	m	1	2	4	4	2	1	2	2	2	5		2	2	35.1	1	2		1	1	1	66	40	3		hypocalcemia		1		hypocalcemic seizure		3	5
82	12456	60	f	1	2	60	60	2	1	2	2	2	10		2	2	44.2	1	2		1	1	1					hyponatremia		1		hyponatremic swizure	mic seizure	2	0
83	12345	100	m	1	2	100	100	1	2	2	2	2	15		2	2	43.1	1	2		1	1	1					hyponatremia		1		hyponatremic swizure	mic seizure	1	0
84	16784	110	m	1	2	110	110	2	2	2	2	2	30		2	2	45.6	1	2		1	1	1					hyponatremia		1		hyponatremic seizure		1	0
85	18532	126	m	2	1	126	126	2	2	2	2	2	12		2	2	40.1	1	2		1	1	1					hyponatremia		2		hyponatremic seizure		2	0
86	15467	96	m	2	1	96	96	2	2	2	2	1	10		2	2	45.4	1	2		1	1	1					hypoglycemia		1		hypoglycemic	mic seizure	4	0
87	1865	84	f	1	2	84	84	2	2	2	2	1	5		2	2	41.2	1	2		1	1	1					hypoglycemia		1		hypoglycemic seizure		4	0
88	19753	75	f	2	2	75	75	1	2	2	2	1	15		2	2	44.3	1	2		1	1	1					hypoglycemia		1		hypoglycemic seizure		4	0
89	12568	37	m	2	1	37	37	2	1	2	2	1	20		2	2	48.5	1	2		1	2	1							1		febrile sd		1	4
90	15374	72	f	1	2	72	72	2	1	2	2	1	5		2	2	49.4	1	2		1	2	1							1		sd		1	4
91	12468	66	m	1	2	66	66	2	2	2	1	1	15		2	2	41.4	1	2		1	2	1							1		sd		1	4
92	14694	40	m	1	2	40	40	1	2	2	2	1	15		2	2	44.6	1	2		1	2	1							1		febrile sd		2	4

93	1423	38	f	1	2	38	38	1	2	2	2	1	25		2	2	47.1	1	2		1	2	1							1		febrile sd		2	4
94	1865	44	m	1	2	44	44	1	2	1	2	2	20		2	2	45.6	1	2		1	2	1							2		sd		2	4
95	14338	54	m	1	2	54	54	2	2	2	1	1	5		2	2	46.5	1	2		1	2	1							2		sd		1	4
96	1823	67	m	1	2	67	67	2	2	2	2	2	45		1	1	46.1	1	1		2	2	1					cp		2		sd		3	3,4&5
97	12267	70	f	2	1	70	70	2	2	1	2	1	30		2	2	45.4	2	2		2	2	1					cp/mr		2		sd		3	4
98	15475	40	m	1	2	40	40	2	2	2	2	2	45		1	2	44.1	2	2		1	2	1					cp		1		sd		3	3&4
99	12975	43	m	2	1	43	43	2	1	2	1	1	15		2	2	43.2	1	2		1	2	1							1		febrile sd		1	4
100	12098	46	m	1	2	46	46	2	1	1	2	1	10		2	2	41.2	2	2		1	2	1							1		febrile sd		1	4

101	1269	52	m	1	2	52	52	2	1	2	2	1	5		2	2	48.4	1	2		1	2	1							1		sd		2	4
102	12956	50	m	1	2	50	50	2	1	2	2	1	25		2	2	49.1	1	2		1	2	1							1		sd		2	4
103	12095	52	f	1	2	52	52	2	1	2	2	1	15		2	2	47.5	1	2		1	2	1							1		sd		2	4
104	11345	120	m	2	1	120	120	1	2	2	2	2	25		2	2	50.2	2	2		2	2	1							1		intracranial tumor		2	4
105	13456	8	m	1	2	8	8	2	1	1	2	1	5		2	2	39.8	2	2		1	2	1							1		febrile sd		1	2
106	13567	74	m	1	2	40	40	2	1	1	1	1	20		2	1	51.2	2	2		1	2	1							2		sd		2	4
107	13678	32	m	1	2	32	32	1	2	2	2	1	25		2	2	49.2	2	2		1	2	1							1		febrile sd		2	4
108	13789	36	m	1	2	36	36	2	1	2	2	1	30		2	2	44.3	2	2		1	2	1							1		febrile sd		2	4
109	13890	142	f	2	1	40	40	2	1	1	2	2	15		2	2	50.1	2	2		1	2	1							1		sd		2	4
110	13901	12	f	1	2	12	12	2	1	2	2	1	20		2	2	42.1	2	2		1	2	1							1		febrile sd		2	4
111	12304	65	m	1	2	42	42	2	2	2	1	3	20		2	2	39.8	2	2		1	2	1							1		sd		2	4
112	12456	136	m	1	2	44	44	2	1	1	1	2	5		2	2	55.1	2	2		1	2	1							1		sd		2	4
113	12567	76	f	2	1	34	34	1	1	2	1	2	12		2	2	50.1	2	2		1	2	1							2		sd		1	4
114	12678	10	m	1	2	10	10	2	2	2	2	1	5		2	2	39.2	2	2		1	2	1							2		febrile sd		1	2
115	12789	40	m	2	1	15	15	1	1	2	1	2	10		2	2	48.5	2	2		1	2	1							1		sd		2	4
116	12890	11	m	1	2	20	20	2	1	2	2	3	20		2	2	40.1	2	2		1	2	1							2		febrile sd		1	2
117	12901	10	f	1	2	10	10	1	2	2	2	1	5		2	2	38.9	2	2		1	2	1							1		febrile sd		2	2
118	14567	73	f	1	2	12	12	2	2	2	1	1	10		2	2	52.2	2	2		1	2	1							1		sd		1	4

119	14678	134	m	1	2	30	30	2	2	2	1	1	10		2	2	53.2	2	2		1	2	1							1		sd		2	4
120	14789	12	m	1	2	12	12	2	2	2	2	1	10		2	2	40.1	2	2		1	2	1							1		febrile sd		1	4
121	14890	38	f	2	1	20	20	2	2	2	1	1	25		2	2	51.1	2	2		1	2	1							2		febrile sd		1	4
122	14901	15	f	2	1	15	15	2	2	2	2	1	15		2	2	44.1	2	2		1	2	1							1		febrile sd		2	4
123	15678	5	m	1	2	5	5	2	1	2	2	2	5		1	2	38.1	2	2		1	2	1							1		febrile sd		2	2
124	15789	17	m	1	2	10	10	2	1	2	1	1	10		2	2	47.1	2	2		1	2	1							1		febrile sd		1	4
125	15890	39	m	1	2	15	15	2	1	2	1	1	25		2	2	49.9	2	2		1	2	1							1		febrile sd		1	4
126	15901	14	m	1	2	14	14	2	2	2	2	1	20		2	2	45.5	2	2		1	2	1							1		febrile sd		2	4
127	15012	132	m	1	2	30	30	2	2	2	1	3	15		2	1	54.5	2	2		1	2	1							1		sd		1	4
128	16789	6	m	1	2	6	6	1	2	1	2	1	20		2	2	43.4	2	2		1	2	1							1		febrile sd		1	2
129	16890	18	m	1	2	18	18	2	2	1	2	1	15		2	2	47.6	2	2		1	2	1							1		febrile sd		1	4
130	16901	40	m	1	2	12	12	2	2	2	1	3	12		2	2	50.1	2	2		1	2	1							1		febrile sd		1	4
131	16012	72	m	1	2	7	7	2	2	2	1	1	16		1	2	52.4	2	2		1	2	1							1		sd		2	4
132	10123	8	m	2	1	8	8	2	1	1	2	1	5		2	2	40.2	2	2		1	2	1							1		febrile sd		1	2
133	10234	20	m	2	1	12	12	2	2	2	1	1	5		2	2	45.5	2	2		1	2	1							1		febrile sd		1	4
134	10345	42	m	1	2	18	18	2	1	1	1	4	5		2	2	42.4	2	2		1	2	1							1		febrile sd		1	4
135	10456	75	m	1	2	20	20	1	2	2	1	4	18		2	1	50.1	2	2		1	2	1							1		sd		2	4
136	10567	80	m	1	2	34	34	1	1	2	1	4	20		2	2	52.1	2	2		1	2	1							1		sd		1	4
137	10678	96	m	1	2	12	12	1	2	2	1	1	15		2	2	53.2	2	2		1	2	1							1		sd		1	4
138	10789	124	m	1	2	18	18	2	2	2	1	4	20		2	2	50.6	2	2		1	2	1							1		sd		1	4
139	10765	76	m	1	2	20	20	1	2	2	1	4	15		2	2	54.4	2	2		1	2	1							1		sd		2	4
140	11023	7	m	2	1	7	7	2	2	2	2	1	10		2	2	42.2	2	2		1	2	1							1		febrile sd		1	2
141	11034	19	m	2	1	19	19	1	2	2	2	1	20		2	2	45.8	2	2		1	2	1							1		febrile sd		1	4
142	11044	42	m	2	1	20	20	2	2	1	1	1	25		2	2	47.2	2	2		1	2	1							1		febrile sd		1	4
143	11054	74	m	2	1	32	32	1	2	2	1	1	15		2	2	49.2	2	2		1	2	1							1		sd		2	4
144	11056	6	m	1	2	6	6	1	1	2	2	1	10		2	2	36.8	2	2		1	2	1							1		febrile sd		1	2
145	11074	16	m	1	2	16	16	1	1	2	2	1	25		2	1	42.1	2	2		1	2	1							1		febrile sd		2	4
146	11084	38	m	1	2	20	20	1	1	2	1	1	25		2	2	50.1	2	2		1	2	1							1		febrile sd		1	4

147	11094	80	m	1	2	33	33	1	2	2	1	1	5		2	2	51.2	2	2		1	2	1							1		sd		1	4
148	12056	7	m	2	1	7	7	2	2	2	2	3	15		2	2	40.3	2	2		1	2	1							1		febrile sd		1	2
149	12076	18	m	1	2	18	18	1	2	2	2	3	20		2	2	48.9	2	2		1	2	1							1		febrile sd		2	4
150	12087	40	m	1	2	36	36	2	2	2	1	3	6		2	2	52.1	2	2		1	2	1							1		febrile sd		2	4

151	12076	88	m	1	2	38	38	2	2	1	1	2	5		2	2	51.2	2	2		1	2	1							1		sd		2	4
152	12097	120	m	1	2	32	32	2	1	2	1	4	10		2	2	54.5	2	2		1	2	1							1		sd		1	4
153	12098	132	m	2	1	48	48	2	1	2	1	4	12		2	2	53.2	2	2		1	2	1							1		sd		1	4
154	12054	124	m	2	1	46	46	2	1	2	1	1	15		2	2	54.1	2	2		1	2	1							1		sd		1	4
155	12076	9	m	2	1	9	9	1	1	1	2	1	5		2	2	38.4	2	2		1	2	1							1		febrile sd		1	2
156	12089	18	m	1	2	18	18	2	1	2	2	1	15		2	2	45.2	2	2		1	2	1							1		febrile sd		1	4
157	12087	42	m	1	2	36	36	2	1	2	1	2	25		2	2	48.2	2	2		1	2	1							1		febrile sd		1	4
158	12095	89	m	1	2	28	29	2	1	2	1	1	10		2	2	51.2	2	2		1	2	1							1		sd		1	4
159	12095	76	m	1	2	20	20	2	1	2	1	2	5		2	2	52.1	2	2		1	2	1							1		sd		2	4
160	12054	140	m	1	2	24	24	2	2	1	1	1	25		2	2	54.2	2	2		1	2	1							1		sd		1	4
161	12067	134	m	1	2	36	36	2	2	1	1	3	15		2	2	53.6	2	2		1	2	1							1		sd		1	4
162	13054	124	m	2	1	48	48	2	2	1	1	1	10		2	1	50.7	2	2		1	2	1							1		sd		1	4
163	14502	10	m	2	1	10	10	2	2	2	2	1	10		2	2	42.3	2	2		1	2	1							1		febrile sd		1	2
164	16984	19	m	1	2	19	19	2	2	2	2	4	25		2	2	47.1	2	2		1	2	1							1		febrile sd		2	4
165	15095	43	m	2	1	24	24	1	2	2	1	1	15		2	2	50.2	2	2		1	2	1							1		febrile sd		1	4
166	15023	90	m	1	2	36	36	1	2	2	1	1	10		2	2	52.1	2	2		1	2	1							1		sd		1	4
167	15024	78	m	2	1	42	42	2	1	2	1	1	5		2	2	51.9	2	2		1	2	1							1		sd		1	4
168	15025	142	m	1	2	48	48	1	1	2	1	1	25		2	2	53.2	2	2		1	2	1							1		sd		1	4
169	15026	136	m	2	1	36	36	2	1	2	1	2	20		2	2	52.2	2	2		1	2	1							1		sd		1	4
170	15027	125	m	2	1	32	32	2	1	2	1	1	15		2	2	50.3	2	2		1	2	1							1		sd		1	4
171	15076	11	m	2	1	11	11	2	1	2	2	1	5		2	2	47.2	2	2		1	2	1							1		febrile sd		2	2
172	15084	18	f	2	1	18	18	2	1	2	1	1	10		2	2	48.2	2	2		1	2	1							1		febrile sd		2	4

173	15034	34	m	1	2	32	32	2	1	2	1	1	10		2	2	50.1	2	2		1	2	1							1		febrile sd		1	4
174	15035	92	m	1	2	26	26	1	1	2	1	2	5		2	2	51.2	2	2		1	2	1							1		sd		2	4
175	15036	80	m	1	2	20	20	1	2	1	1	1	8		2	2	52.1	2	2		1	2	1							1		sd		1	4
176	15037	144	m	1	2	48	48	1	2	2	1	1	10		2	2	54.5	2	2		1	2	1							1		sd		1	4
177	15038	130	m	1	2	44	44	1	2	2	1	1	10		2	2	53.2	2	2		1	2	1							1		sd		1	4
178	15039	122	m	1	2	36	36	1	2	2	1	3	10		2	2	51.7	2	2		1	2	1							1		sd		1	4
179	16054	96	m	1	2	40	40	2	2	2	1	1	16		2	2	52.2	2	2		1	2	1							1		sd		1	4
180	15011	62	m	2	1	24	24	1	1	2	1	1	20		2	2	51.8	2	2		1	2	1							1		sd		2	4
181	15015	73	m	2	1	36	36	2	1	1	1	1	25		2	2	52.4	2	2		1	2	1							1		sd		2	4
182	15017	84	m	2	1	32	32	2	1	2	1	1	10		2	2	53.3	2	2		1	2	1							1		sd		2	4
183	15019	95	f	2	1	37	37	2	1	2	1	2	14		2	2	51.2	2	2		1	2	1							1		sd		3	4
184	15020	106	m	1	2	38	38	2	2	2	1	1	15		2	2	52.2	2	2		1	2	1							1		sd		2	4
185	15064	117	m	1	2	25	25	2	2	2	1	1	10		2	2	51.9	2	2		1	2	1							1		sd		2	4
186	12876	128	m	1	2	26	26	1	2	2	1	1	5		2	2	50.9	2	2		1	2	1							1		sd		3	4
187	13986	139	f	1	2	28	28	1	2	2	1	1	5		2	2	53.2	2	2		1	2	1							1		sd		3	4
188	15065	138	m	1	2	30	30	1	2	2	1	1	5		2	2	52.8	2	2		1	2	1							1		sd		2	4
189	15043	127	m	1	2	32	32	1	1	2	1	1	15		2	2	53.1	2	2		1	2	1							1		sd		2	4
190	15098	116	m	1	2	12	12	2	2	2	1	1	20		2	2	51.8	2	2		1	2	1							1		sd		2	4
191	16054	107	f	1	2	18	18	2	1	2	1	2	10		2	2	52.6	2	2		1	2	1							1		sd		2	4
192	1468	94	f	1	2	32	32	2	1	2	1	3	15		2	2	53.4	2	2		1	2	1							1		sd		2	4
193	16492	83	f	1	2	36	36	2	2	1	1	1	15		2	2	53.6	2	2		1	2	1							1		sd		2	4
194	13804	72	m	2	1	54	54	2	2	1	1	1	10		2	2	52.8	2	2		1	2	1							1		sd		2	4
195	15047	61	m	1	2	48	48	2	2	2	1	1	25		2	2	51.9	2	2		1	2	1							1		sd		1	4
196	13402	65	f	1	2	46	46	1	2	2	1	1	25		2	2	52.8	2	2		1	2	1							2		sd		3	4&5
197	184452	76	m	1	2	34	34	2	1	2	1	1	10		2	2	53.7	2	2		1	2	1							2		sd		3	4&3
198	19552	87	f	1	2	27	27	1	2	1	1	1	10		2	2	54.7	2	2		1	2	1							1		sd		2	4
199	18452	98	m	1	2	42	42	2	2	2	1	3	15		2	2	52.7	2	2		1	2	1							2		sd		2	4
200	17452	109	f	1	2	37	37	2	1	2	1	2	10		2	2	53.6	2	2		1	2	1							1		sd		2	4

201	18462	111	m	1	2	38	38	2	1	2	1	2	5		2	2	54.9	2	2		1	2	1							1		sd		2	4
202	19442	124	m	2	1	30	30	2	1	2	1	1	5		2	2	53.8	2	2		1	2	1							1		sd		2	4
203	12330	136	m	2	1	30	30	2	1	2	1	1	5		2	2	54.7	2	2		1	2	1							2		sd		2	4
204	17452	66	f	2	1	42	42	2	1	2	1	1	25		2	2	53.9	2	2		1	2	1							1		sd		2	4
205	13294	78	f	2	1	40	40	2	2	1	1	3	20		2	2	52.6	2	2		1	2	1							2		sd		2	4
206	15239	88	m	2	1	22	22	1	2	2	1	1	15		2	2	52.8	2	2		1	2	1							1		sd		2	4
207	18252	90	f	1	2	32	32	1	2	2	1	1	5		2	2	53.5	2	2		1	2	1							1		sd		1	4
208	15230	112	f	1	2	24	24	1	2	2	1	1	12		2	2	52.5	2	2		1	2	1							1		sd		1	4
209	15284	124	m	1	2	48	48	2	1	2	1	1	10		2	2	54.9	2	2		1	2	1							1		sd		2	4
210	12094	138	f	1	2	36	36	2	1	2	1	1	15		2	2	52.8	2	2		1	2	1							1		sd		2	4

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DISSERTATION SUBMITTED FOR THE DEGREE OF

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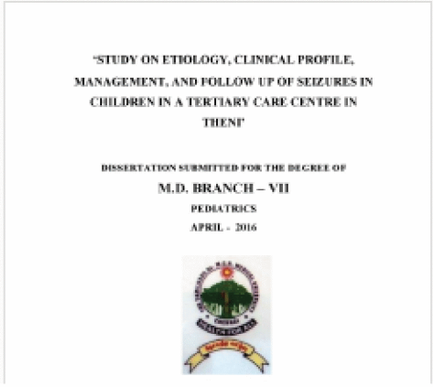

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